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(54) Title: METHODS AND COMPOSITIONS FOR TREATING NEUROBEHAVIORAL DISORDERS

(57) Abstract: This composition is for treating neurobehavioral disorders, by restoring normal neurotransmitter, receptor, transport and metabolic function. The first stage of treatment is to administer a composition intravenously designed to treat the patient's symptoms. The next stage is supplemental oral support. This invention embodies compositions for intravenous treatment of certain types of neurobiological disorders and methods of diagnosis, which comprises specialized testing and pre-diagnosis of underlying neurological conditions, immunization, and methods of education and psychological support available remotely through the Internet or by mail.



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METHODS AND COMPOSITIONS FOR TREATING NEUROBEHAVIORAL DISORDERS

Related Cases:

5 This application claims priority to United States Provisional Patent
Application Serial No.60/158,604 titled "Method of Treating Reward Deficiency
Syndrome Disorders" Joyce C. "Kim" Bechthold, inventor, filed October 8,
1999; United States Provisional Patent Application Serial No: 60/164,049, titled
"Method for Treating Reward Deficiency Syndrome Disorders", filed November
10 8, 1999, Joyce Corinne Bechthold, inventor; United States Provisional Patent
Application Serial No: 60/166,068, filed November 17, 1999, titled "Method for
Treating Reward Deficiency Syndrome Disorders," Joyce Corinne Bechthold,
inventor; and United States Provisional Patent Application Serial No:
60/201,043, titled "Method for Treating Reward Deficiency Syndrome
15 Disorders", Joyce Corinne Bechthold, inventor, filed May 1, 2000. Each of the
above Provisional Patent Applications is herein incorporated fully by reference.

BACKGROUND

20 I. Field of the Invention

 This invention relates to compositions and methods useful for treating
central nervous system disorders. Specifically, the compositions comprise one or
more amino acids, neurotransmitter precursors, vitamins, minerals, enzyme
inhibitors, corticosteroids and/or immunological enhancers, and also comprises
25 methods for intravenous administration of those compositions.

II. Description of Related Art

Neurochemical pathways in the brain are associated with behavioral phenomena. Seeking behaviors involve pathways that have been termed “reward pathways,” and such pathways in the brain have been known since they were reported in 1954, and are implicated in drives for reproduction, eating and other pleasurable activities (for a review, see Blum et al. American Scientist 84:132-146 (1996) and Noble, Alcohol 16(1):33-45 (1998), both incorporated herein fully by reference). Abnormalities in these neurochemical pathways, however, can result in disorders, herein termed “neurobehavioral disorders.” Among these disorders, addictions to alcohol, nicotine and other drugs continue to be a public health problem. Additionally, numerous psychological conditions including obsessive compulsive behavior and attention deficit hyperactivity disorder have been associated with certain abnormalities in neurochemical pathways.

Alcoholism affects one in 20 individuals in the United States and just under one in 20 worldwide. Drug addiction affects one in every 100 individuals in the United States and just under 1 in 100 individuals worldwide. Nearly four million individuals enter treatment for drug and/or alcohol addiction in the United States every year. It is estimated that only 10 to 12% of those currently entering conventional treatment will not relapse. The cost to society of addictive disorders is about \$20 billion yearly.

Physiological Basis for Neurobehavioral Disorders

The underlying biochemistry of addictive and/or compulsive behaviors has been a subject of study for many years. However, biological processes involved in neurobehavioral disorders have, until recently, been poorly understood.

One widely studied theory is based upon neurobiological mechanisms, wherein abnormalities of neurotransmitter release, neurotransmitter receptors, and/or neurotransmitter degradation may be associated with neurobehavioral

disorders. The regulation of certain biogenic amine neurotransmitters is altered in a variety of psychiatric and neurobehavioral disorders. There are five biogenic amine neurotransmitters: three catecholamines, norepinephrine (noradrenaline), epinephrine (adrenaline) and dopamine, histamine and serotonin. Drugs that alter
5 bioamine neurotransmission are common psychiatric medications in use today.

There are correlations between functioning of the adrenergic nervous system; the dopaminergic nervous system, which uses dopamine as the transmitter and dopaminergic receptors; the serotonergic nervous system, which uses serotonin as a transmitter; and the GABA-ergic system, which uses gamma-
10 amino butyric acid ("GABA") as a transmitter. These neurobiological systems play significant roles in behaviors relating to addiction to alcohol, cocaine, heroin, morphine, other chemicals, and to foods.

These transmitters are biochemically synthesized from amino acid precursors. For example, the adrenergic neurotransmitter norepinephrine can be
15 synthesized from L-tyrosine, dopamine can be synthesized from L-tyrosine, L-phenylalanine, and L-DOPA. Serotonin can be biochemically synthesized from L-tryptophan and 5-hydroxytryptophan, and GABA can be synthesized from L-glutamine, L-glutamate, and L-glutamic acid.

Most psychotropic drugs (defined as drugs that alter behavior, mood or
20 perception) selectively affect one or more steps in the synthesis, packaging or degradation of biogenic amines. There are several broad categories of psychotropic drugs: anti-psychotics, anti-anxiety drugs, antidepressants and stimulants. These drugs, except for the benzodiazepines (e.g., Valium™, etc.) generally affect aminergic transmission. Drugs such as Prozac™, trazadone and
25 desipramine specifically affect aminergic transmission, involving, for example, serotonin pathways. Stimulants such as amphetamines can cause the release of norepinephrine and other catecholamines from nerve terminals and increase the effects of released catecholamines. Despite the relatively small number of

aminergic neurons in the brain, a litany of pharmacological reactions involved with them suggest that these neurons are critically important in the maintenance of health.

Behavioral changes can occur in animals following systemic and direct
5 central nervous system delivery of precursor amino acids. Certain L-amino acids can be neurotransmitter and neuromodulator precursors. Additionally, D-amino acids such as D-phenylalanine and D-leucine can decrease the degradation of opioid peptides that are central to regulation of mood. Unless otherwise stated, the term "amino acids" as used herein refers to L-amino acids, D-amino acids, or
10 DL-amino acids.

Many transmitters involved in the nervous system are synthesized from amino acid precursors. Thus, the amounts of precursor amino acids present in an individual can influence the production of the transmitters and thereby affect physiological functions of the systems that use those transmitters. In fact, amino
15 acid precursors and inhibitors of opioid peptide degradation can have therapeutic effects on patients suffering from addictive and compulsive disorders.

20 **Structure-Biochemical-Function Relationships in Neurobehavioral Disorders**

Several brain structures can be involved in neurobehavioral disorders. According to one theory, neurobiological pathways associated with seeking behaviors include the dopaminergic nervous system, the ventral tegmental area (VTA), the nucleus accumbens, the olfactory tubercle, the ventral striatum and
25 the frontal cortex. Also, according to this theory, the release of dopamine near receptors in the hippocampus and nucleus accumbens can be of significant importance in addictive and compulsive disorders.

Certain conditions such as attention deficit hyperactivity disorder (ADHD), depression, anxiety, panic disorder, compulsive sexual activity, pathological gambling, and compulsively engaging in high-risk activities can involve neurophysiological mechanisms.

5 Certain pharmaceutical agents can affect neurotransmitter levels in the limbic nervous system and in higher centers, including the cerebrum. The limbic system includes deep brain structures including the amygdala, nucleus accumbens, and the hippocampus. In one group of studies (Amen et al., Primary Psychiatry, August 1998), incorporated herein fully by reference, cocaine users
10 showed regions of significant cerebral hypoperfusion in the frontal, paraventricular, and/or temporal-parietal areas of their brains. Deficits in attention, concentration, new learning, visual and verbal memory, word production, and visuomotor integration were observed. Crack cocaine users showed a 23% decrease in cerebral blood flow compared to controls, and crack
15 cocaine users who were also cigarette smokers showed a 42% decrease in cerebral blood flow overall compared to controls. The study indicated that long-term cocaine use might produce sustained brain perfusion deficits and persistent neuropsychological compromise in some groups of cocaine-using patients.

 In another study, a genetic vulnerability to alcohol is suspected because
20 SPECT abnormalities are more frequent in patients with a family history of drinking problems. After abstinence, abnormalities seen on SPECT imaging only partially resolved. Acute and chronic substance use appears to affect cerebral blood flow and metabolism, thus compromising central nervous system functioning. This seems to be especially true in the areas of the frontal lobes,
25 which can control executive functioning, and in the temporal lobes, which are involved in mood stability and aggression on the dominant side.

Genetic Association of Neurobehavioral Disorders

Some correlations exist between neurobehavioral disorders and underlying genetic conditions. For example, the dopamine D₂ receptor gene is associated with the appearance of compulsive disorders in human beings (Blum et al., U.S. Patent No: 5,500,343, incorporated herein fully by reference. With the breeding of the alcohol-preferring D57 strain of mice through successive generations, a genetic basis for alcoholism was developed. The association of dopamine receptors and alcoholism has also been established. The dopamine D2 receptor gene has four known Taq1A alleles, the A1, A2, A3, and the A4. The A3 and A4 are rare, the A2 is found in about 75% of the general population and the A1 is found in about 25%. Sixty-nine percent of alcoholics in one study had the A1 allele and 20% of non-alcoholics had the A1 allele. Additionally, 14 independent laboratories have supported the finding that the A1 allele is associated with severe forms of alcoholism, though perhaps not in milder forms (Blum and Noble, Science 265:1346-1347 (1994), incorporated herein fully by reference).

Additionally, there is an association between the A1 allele and a prolonged latency of the P300 wave in the electroencephalogram (EEG) in children of alcoholics. D2 receptors are decreased by chronic cocaine administration, and this may induce craving for cocaine. Moreover, childhood behavioral disorders may signal a genetic predisposition to drug or alcohol addiction. In a population of males with problems of drug addiction who were also pathological gamblers, the incidence of the A1 allele rose to 76 percent. Moreover, in a study of identical twins, if one twin had ADHD there was a 100% probability that the other twin also had the disorder. One study found that 59% of Vietnam veterans with post-traumatic stress disorder carried the A1 allele as opposed to only 5% who carried the allele, and were exposed to similar stress but did not develop the disorder. Positron emission tomography (PET) scan studies

have been reported to show that neurons of individuals carrying the A1 allele have approximately 30% fewer D2 receptors than those carrying the A2 allele. In particular, the A₁ allele has been associated with Tourette's Syndrome, ADHD, autism, post-traumatic stress disorder, pathological gambling, smoking
5 and obesity

Current Therapeutic Approaches to Neurobehavioral Disorders

Currently, addictive disorders are treated primarily by psychosocial intervention with adjunct therapy with certain orally administered pharmaceutical
10 drugs. Certain drugs, such as Valium™, are prescribed to aid in withdrawal, and others, such as naloxone and acamprosate, are being prescribed for and/or are being studied to aid in relapse prevention. Several amino acid formulations used as oral supplements are available as an aid in curbing craving, attention deficit hyperactivity disorders (ADHD), some psychiatric disorders and weight gain
15 (Blum et al. U.S. Patent 5,550,021, incorporated herein fully by reference). Most neurobehavioral disorders are treated with single pharmaceutical medications and/or psychiatric counseling.

Currently, patients with addictive disorders are being treated without first being diagnosed, or are being treated for underlying neurological deficits, injury
20 related to substance use, attentional disorders or brain trauma. Certain individuals seek and self-administer addictive substances and engage in certain activities to attempt to restore a more normal functioning of the neurotransmitters in their nervous systems.

However, patients may not be able to maintain their recovery from
25 addiction or other disorders if underlying functional physiological and biochemical deficits or brain traumas remain untreated. Further, the neurological damage of drug and alcohol use can itself cause poor executive function and decision making. If both functional deficits and brain trauma remain unresolved

prior to treatment, such patients are at risk of relapsing. Currently, especially in treatment of drug and alcohol addiction, large numbers of patients fail to recover, which suggests that an underlying factor is being missed.

5 Fear of experiencing withdrawal can discourage patients from seeking, carrying out and completing therapies for addiction. Withdrawal can be characterized by presence of undesirable physiological and psychological symptoms that can include pain, disphoria and muscle tremors, which can include delerium tremens. Additionally, the patient can experience continual cravings and the fear of imminent relapse. There are currently no effective pharmaceutical
10 treatments that can eliminate or significantly reduce the symptoms of withdrawal and prevents relapse over the long term.

 Several pharmaceutical drugs appear to slightly reduce cravings in patients having addictive disorders. An oral nutritional supplement including amino acids, vitamins, and selected minerals appears to mildly reduce craving.
15

SUMMARY OF THE INVENTION

 Thus, one object of this invention is the development of compositions that can minimize adverse effects of addiction and other neurobehavioral disorders in patients recovering from said disorders.

20 Another object of this invention is the development of methods of administrating compositions that improve the therapeutic efficacy of compositions used to treat addiction and other neurobehavioral disorders.

 Another object of this invention is the diagnostic evaluation of neurobiological conditions of patients having neurobehavioral disorders, and the
25 formulation of compositions designed to treat those specific conditions.

 To meet these and other objectives, it was discovered that intravenously administered compositions that contain neurotransmitter precursors, amino acids, vitamins, inhibitors of neurotransmitter degradation and/or immune function

enhancers resulted in unexpected and dramatic reduction in symptoms associated with withdrawal, improvement in symptoms of drug and alcohol overuse and reduction or cessation of cravings for addictive substances. These compositions and methods can permit the brain to function more normally, and can eliminate or decrease symptoms of withdrawal, craving and compulsion associated with addiction and other central neurobiological disorders. These effects can result in longer lasting improvement in symptoms, thereby reducing the risk of relapse. These effects can also result in the patient completing treatment because the ill effects of withdrawal are reduced.

In certain aspects of this invention, compositions comprise amino acid precursors of neurotransmitters in amounts sufficient to relieve craving and other symptoms of addictive states.

In other aspects of this invention, inhibitors of neurotransmitter degradation can be added to neurotransmitter precursors to support or increase the function of neurochemical pathways that are deficient in the disorder.

In yet other aspects of this invention, corticosteroids, gamma-globulins and/or other immune system enhancers and hormones can improve the efficacy of neurotransmitter precursors, amino acids and inhibitors of neurotransmitter degradation.

Other embodiments of this invention include compositions comprising amino acids and other substances to be taken orally to support normal neurotransmitter function attained by intravenous administration.

Yet other embodiments of this invention include methods for treating distinct subsets of patients with neurobehavioral disorders.

Other embodiments of this invention include methods of diagnosis and/or evaluation of underlying neurobehavioral disorders, along with reevaluation during the course of therapy. Certain of these methods comprise different technologies, review of the patient's history, and/or written questionnaires.

Certain diagnostic and brain imaging methods include: (1) single photon emission computed tomography (SPECT), (2) quantitative electroencephalography (qEEG) and (3) infrared spectrophotometry.

Additional evaluative methods that can be useful to the practice of the methods of this invention can include analysis of thyroid function, presence of genetic indicators of neurobehavioral disorders, presence of amino acid deficiencies, metabolic impairments, nutritional deficiencies, food and chemical sensitivities and amino acid transport disorders. These diagnostic and predictive tests can permit design of individualized intravenous and oral compositions and methods of treatment. For example, as described further below, there are known associations of certain types of neurological and physiological characteristics with results of diagnostic and evaluative procedures. By measuring a patient's condition during a course of therapy, the efficacy of the treatment can be evaluated and compositions and routes of administration can be altered if so desired.

Another aspect of this invention includes education of the patient and patient's family, and psychosocial support. Such support and information can be accessible remotely through the internet or computer, and can include ongoing communication of patient information and test results, as part of the treatment and follow-up.

DETAILED DESCRIPTION OF THE INVENTION

To practice this invention, key neurotransmitter precursors, enzyme inhibitors, antiinflammatory agents, amino acids, vitamins and/or other pharmaceutically active agents can be infused directly into the blood stream of a patient affected by neurobehavioral disorders. The injected substances can result in the improvement or normalization of activity in the brain. These therapies can reduce or eliminate the symptoms of withdrawal and continued craving for the

addicting substance, and can prevent or delay the onset of the disorders. In subjects with non-addictive neurobehavioral disorders, similar approaches can normalize brain function and can thereby lessen or abate the adverse symptoms of those disorders.

5 Alternative routes of administration of therapeutic agents are provided that can provide an adjunct to the intravenous therapies. Methods of evaluating the efficacy of treatment are provided to give the patient, family and others the information necessary to understand the basis of the disorder, follow and evaluate treatment efficacy and better predict and prevent relapse, and to provide rapid
10 responses to a patient's needs.

 A significant advantage of compositions of this invention is that they can be less expensive compared to current costs of residential treatment for drug and alcohol addiction. Further, because of the high rate of relapse of the present methods, costs incurred to repeat therapy can be reduced. This can be especially
15 true in situations in which residential care and/or hospitalization would otherwise be required.

 When patients achieve a feeling of well-being and are sufficiently clear headed they can better maintain their recovery. In contrast, prior art methods of treating addiction using oral and/or psychosocial methods can result in
20 inconsistent results and take much longer to be effective if they are effective at all. This may be because the patients may never have been truly well, at ease or clear-headed.

 Another surprising discovery is that using intravenous delivery, many addicted patients can experience reduced or no withdrawal symptoms.

25 A substantial fear among many addicts is the knowledge of how bad they feel when they do not have their drug of choice. Such fear can keep addicts from entering into treatment. Because the treatments of this invention can be easy to undergo and are effective, persons who are reticent can be more willing to enter

treatment. If addicted people feel better immediately, and continue to feel better each successive day of treatment, the patients may be more willing to remain in treatment once they start, even without making a major commitment in advance to a long-term course of therapy.

5 Another advantage of the intravenous methods of this invention is that is not be necessary for patients to enter a residential facility to receive treatment.

 Another benefit of intravenous methods of this invention is the change in attitude toward the patients and toward addictive disorders that the treatment can foster. Addiction is widely viewed as a result of a character or spiritual
10 deficiency, and addicts are commonly stigmatized. A view prevails that if an addict relapses, that addict is lacking the will to succeed. Educating the patient, the patient's family, and society at large in the neurobiological basis for behavioral disorders can be an impetus to recovery, not only for the patient, but also for the family and for society as a whole.

15 A premise of the present invention is that addiction, brain trauma and other forms of neurobehavioral disorders are in large part physiological, having causes and mechanisms that can be understood in physiological terms. Traditional psychological adjuncts can be supportive of the methods of this invention. The fact that the addictive disorders can be successfully treated
20 medically, not behaviorally, can cause public perception about behavioral disorders to change, and the negative stigmata can be reduced, thereby improving the prospects of recovery and integration into society without fear of job discrimination, social discrimination or any other loss of stature or respect that each person should be accorded.

25 Because the compositions and methods of this invention can be administered by physicians and be approved by the FDA, insurance payors may provide funds for treatment. The cost to society of drug and alcohol addiction in one year is over \$20 billion. However, much of this cost is due to incarceration

rather than treatment of addicts. A medical treatment that works can be welcomed by private insurers, the federal government, the justice system and county and state agencies that pay for treatment for their constituents.

Finally, certain embodiments of this invention can be useful for disorders
5 involving carbohydrate addiction and weight gain, nicotine addiction and disorders characterized by potentially life-threatening, risk-taking activities. For example, weight gain can be treated more efficiently when craving is initially decreased using intravenous administration methods of this invention. In the case
of patients experiencing depression, anger and/or hostility, the intravenous
10 administration of compounds of this invention can be life saving. In particular, the intravenous administration of amino acids can relieve the symptoms in patients with severe depression and suicidal ideation.

In patients having attention deficit hyperactivity disorders (ADHD), heightened levels of awareness and attention and relief of anxiety can occur
15 immediately with intravenous administration of compositions of this invention. This improvement can encourage the patient to understand that ADHD is physiological in nature and not a result of lack of intelligence or character. The patient can thus be encouraged to continue treatment.

20 I. Neurobehavioral Disorders

The term "neurobehavioral disorders" as used herein is defined as a constellation of symptoms involving biochemical and physiological neurotransmitter mechanisms in the brain that normally provide what we consider to be normal neurochemical function, " sometimes called by others "reward."
25 The term "reward" is more limiting than the term "normal neurochemical function," for simplicity, we will use the term "normal function" herein to mean normal neurochemical function. The neurotransmitters involved in the biology of normal function is complex; several neurotransmitters can be involved in

promoting normal function at several sites in the brain: dopamine, acting on D₂ receptors, serotonin acting in the hypothalamus, the enkephalins (opioid peptides) in the ventral tegmental area in the nucleus accumbens, and the inhibitory neurotransmitter GABA in the ventral tegmental area and the nucleus accumbens.

Neurobehavioral disorders include but are not limited to obesity, smoking, Tourette's Syndrome, ADHD, ADD, Schizoid/Avoidant Behavior, aggression, posttraumatic stress syndrome, alcoholism, drug addiction, obsessive compulsive behaviors, learning disorders, reading problems, gambling, manic symptoms, phobias, panic attacks, oppositional defiant behavior, conduct disorder, sexual behavior disorders, schizoid disorders, somatization disorders, depression, sleep disorders, general anxiety disorders, stuttering, tic disorders, anger and violent behavior disorders.

Additional examples of neurobehavioral disorders are taken from the Quick Reference to the Diagnostic Criteria From DSM-IV™, The American Psychiatric Association, Washington, D.C., 1994, including but not limited to: Anxiety disorders, include Panic Disorder Without Agoraphobia, 300.01, Panic Disorder With Agoraphobia, 300.21, Agoraphobia Without History of Panic Disorder, 300.22, Specific Phobia, 300.29, Social Phobia, 300.23, Obsessive-Compulsive Disorder, 300.3, Posttraumatic Stress Disorder, 309.81, Acute Stress Disorder, 308.3, Generalized Anxiety Disorder, 300.02, Overanxious Disorder of Childhood, 300.02, Anxiety Disorder Due to [Indicate general medical condition], 293.89, Substance Induced Anxiety Disorder, 293.89, Anxiety Disorder NOS, 300.00; Attention Deficit and Disruptive Behavior Disorders, including Attention-Deficit/Hyperactivity Disorder, Predominately Inattentive Type, 314.00, Attention/Deficit Hyperactivity Disorder, Predominately Hyperactivity-Impulsive Type, 314.01 Attention-Deficit/Hyperactivity Disorder, Combined Type, 314.01,

Attention-Deficit/Hyperactivity Disorder NOS, 314.9, Conduct Disorder, 312.8
Oppositional Defiant Disorder, 313.81, Disruptive Behavior Disorder NOS.,
312.9; Bipolar Disorders including Bipolar I Disorder, 296.0x, 296.40, 296.4x,
296.6x, 296.5x, and 296.7, Bipolar II Disorder, 296.89, Cyclothymic Disorder,
5 301.13, Bipolar Disorder NOS, 296.80; Depressive Disorders including Major
Depressive Disorder, Recurrent, 296.3. Dysthymic Disorder, 300.4, Depressive
Disorder, 311, Major Depressive Disorder. Single Episode, 296.2, Eating
Disorders including Bulimia Nervosa, Nonpurging Type, 307.51, Bulimia
Nervosa, Purging Type, 307.51, Anorexia Nervosa, 307.1, Eating Disorder NOS
10 307.50; Impulse Control Disorders including Intermittent Explosive Disorder,
312.34, Kleptomania, 312.32, Pyromania, 312.23, Pathological Gambling,
312.31. Tricotillomania, 312.39, Impulse Control Disorder NOS, 312.30;
Personality Disorders including Antisocial Personality Disorder, 301.7, Avoidant
Personality Disorder, 301.82, Obsessive-Compulsive Personality Disorder, 301.4
15 Schizoid Personality Disorder, 301.20; Schizophrenia including Paranoid Type.
295.30, Disorganized Type, 295.10, Catatonic Type, 295.20, Undifferentiated
Type, 295-90, Residual Type, 295.60, Schizoaffective Disorder, 295.70,
Schizophreniform Disorder, 295.40; Sleep Disorders including Primary Sleep
Disorders such as Dyssomnias which include Primary Insomnia 307.42, Primary
20 Hypersomnia 307.44, Narcolepsy 347, Circadian Rhythm Sleep Disorder,
307.45, Dyssomnia NOS 307.47, Parasomnias which include Nightmare Disorder
307.47, Sleep Terror Disorder 307.46, Sleepwalking Disorder 307.46,
Parasomnia NOS 307.47, Sleep Disorders Related to Another Mental Disorder
which include Insomnia Related to [indicate Axis I or Axis II disorder] 307.42,
25 Hypersomnia Related to [Indicate Axis I or Axis II disorder] 307.44, Other Sleep
Disorders which include Sleep Disorder Due to [Indicate the General Medical
Condition] 780.xx, Substance Induced Sleep Disorder 780.xx; Substance Use
Disorders including Alcohol Related Disorders such as Alcohol-Induced

Psychotic Disorder, with delusions, 291.5, Alcohol Abuse, 305.00, Alcohol Intoxication, 303.00, Alcohol Withdrawal, 291, Alcohol Intoxication Delirium, 291.0, Alcohol Withdrawal Delirium, 291.0, Alcohol-Induced Persisting Dementia, 291.2, Alcohol-Induced Persisting Amnestic Disorder, 291.1, Alcohol Dependence, 303.90, Alcohol-Induced Psychotic Disorder, with hallucinations, 291.3, Alcohol-induced Mood Disorder, 291.8, Alcohol-induced Anxiety Disorder, 291.8, Alcohol-induced Sexual Dysfunction, 291.8, Alcohol-induced Sleep Disorder, 291.8. Alcohol-Related Disorder NOS, 291.9, Alcohol Intoxication, 303.00, Alcohol Withdrawal, 291.8, Nicotine Related Disorders which include Nicotine Dependence, 305.10, Nicotine Withdrawal, 292.0, Nicotine- Related Disorder NOS, 292.9, Amphetamine Related Disorders which include Amphetamine Dependence, 304.40, Amphetamine Abuse, 305.70, Amphetamine Intoxication, 292.89, Amphetamine Withdrawal, 292.0, Amphetamine Intoxication Delirium, 292.81, Amphetamine-Induced Psychotic Disorder with delusions, 292.11, Amphetamine-Induced Psychotic Disorders with hallucinations, 292.12, Amphetamine no- Induced Mood Disorder, 292.84, Amphetamine-Induced Anxiety Disorder, 292.89. Amphetamine- induced Sexual Dysfunction, 292.89, Amphetamine-Induced Sleep Disorder, 292.89, Amphetamine Related Disorder NOS, 292.9, Amphetamine Intoxication, 292.89, Amphetamine Withdrawal, 292.0. Cannabis Related Disorders which include Cannabis Dependence, 304.30, Cannabis Abuse, 305.20, Cannabis Intoxication, 292.89, Cannabis Intoxication Delirium, 292.81, Cannabis- Induced Psychotic Disorder, with delusions, 292.11, Cannabis-induced Psychotic Disorder with hallucinations, 292.12, Cannabis-induced Anxiety Disorder, 292-89, Cannabis Related Disorder NOS, 292.9, Cannabis Intoxication, 292.89, Cocaine Related Disorders which include Cocaine Dependence, 304.20, Cocaine Abuse, 305.60, Cocaine Intoxication, 292.89, Cocaine Withdrawal, 292.0, Cocaine Intoxication Delirium, 292.81, Cocaine-Induced Psychotic Disorder with delusions, 292.11,

Cocaine-Induced Psychotic Disorders with hallucinations, 292.12, Cocaine-Induced Mood Disorder, 292.84, Cocaine-induced Anxiety Disorder, 292.89, Cocaine-Induced Sexual Dysfunction, 292.89, Cocaine-Induced Sleep Disorder, 292.89, Cocaine Related Disorder NOS, 292.9, Cocaine Intoxication, 292.89, Cocaine Withdrawal, 292.0; Hallucinogen Use Disorders which include Hallucinogen Dependence, 304.50, Hallucinogen Abuse, 305.30, Hallucinogen Intoxication, 292.89, Hallucinogen Withdrawal, 292.0, Hallucinogen Intoxication Delirium, 292.81, Hallucinogen- Induced Psychotic Disorder with delusions, 292.11, Hallucinogen -Induced Psychotic Disorders with hallucinations, 292.12, Hallucinogen-Induced Mood Disorder, 292.84, Hallucinogen-Induced Anxiety Disorder, 292.89, Hallucinogen Induced Sexual Dysfunction, 292.89, Hallucinogen-Induced Sleep Disorder, 292.89, Hallucinogen Related Disorder NOS, 292.9, Hallucinogen Intoxication, 292.89, Hallucinogen Persisting Perception Disorder (Flashbacks), 292.89; Inhalant Related Disorders which include Inhalant Dependence, 304.60, Inhalant Abuse, 305.90, Inhalant Intoxication, 292.89, Inhalant Intoxication Delirium, 292.81, Inhalant-Induced Psychotic Disorder, with delusions, 292.11, Inhalant- Induced Psychotic Disorder with hallucinations, 292.12, Inhalant-Induced Anxiety Disorder, 292.89, Inhalant Related, Disorder NOS, 292.9, Inhalant Intoxication, 292.89; Opioid Related Disorders which include Opioid Dependence, 304.00, Opioid Abuse, 305.50, Opioid Intoxication, 292.89, Opioid Intoxication Delirium, 292.81, Opioid-Induced Psychotic Disorder, with delusions, 292.11, Opioid-Induced Psychotic Disorder with hallucinations, 292.12, Opioid-Induced Anxiety Disorder, 292.89, Opioid Related Disorder NOS, 292.9, Opioid Intoxication, 292.89, Opioid Withdrawal, 292.0; Polysubstance Related Disorders which include Polysubstance Dependence, 304.80; Tic Disorders which include Tourette's Disorder, 307.23, Chronic Motor or Vocal Tic Disorder 307.22, Transient Tic Disorder 307.21, Tic Disorder NOS 307.20, Stuttering 307.0,

Autistic Disorder, 299.00, and Somatization Disorder 300.81. Additionally, other Neurobehavioral disorders are defined as would be known to one of skill in the art, such as Novelty Seeking, defined in (Clonigen *et al.*, 1993). Other disorders, including Huntington's Chorea, Amyotrophic Lateral Sclerosis, Environmental
5 Sensitivity, Chemical Injury Syndrome, Chronic Fatigue Syndrome, and any others, if not specifically defined herein, that are the same as commonly known to one of skill in the art, including common abbreviations.

A. Genetic Basis for Neurobehavioral Disorders

10 Neurobehavioral disorders and addiction can have common genetic components. Neurons in the brains of individuals suffering from addiction to alcohol and drugs can have fewer receptors for the neurotransmitter dopamine. A particular genetic mutation has been shown to be associated with the reduced number of receptors for dopamine on brain neurons. Approximately thirty
15 percent of the population share the genetic mutation. Seventy-one percent of individuals suffering from severe alcohol and drug addiction have this genetic mutation in common.

If neurons in the brain do not have sufficient dopamine activity, brain function can be significantly changed. The change in brain activity can be unique
20 for each person. Some individuals may find it difficult to focus on complex thoughts, some individuals might feel jumpy, some individuals might be anxious or easily irritable. Some children may be unmanageable or consistently in trouble. Some adults may be gambling compulsively, driving recklessly, carbohydrate binging, compulsively thrill seeking or compulsively requiring
25 sexual orgasm.

Persons with less than normal dopamine concentrations may not be "at ease" or "comfortable" without first finding some means to increase dopamine in

their brains. One way to stimulate the production of dopamine can be to drink alcohol, use cocaine or other addictive substances.

For many neurobehavioral disorders, the malfunction can be genetically determined. Disorders caused by head trauma and/or chemical brain injury may be the exceptions, but can be present as a similar constellation of physiological and behavioral traits as do genetically associated disorders, and can be treated similarly, with similar benefits using the compositions and methods of this invention.

10 **B. ADHD and Addictive Disorders**

Addiction and Attention Deficit Hyperactivity Disorder (ADHD) can be genetically related. Dopamine activity can play an important role in both disorders. Children with ADHD have a high risk of becoming addicted to drugs and alcohol (Biederman, et al., Pediatrics 104:2, (August 1999), incorporated
15 herein fully by reference). Moreover, a high percentage of adults who are addicted to drugs and alcohol, particularly those who are addicted to cocaine, were not treated for ADHD as children when they clearly had the disorder. Although the causes of developing cocaine addiction are not completely known, one hypothesis is that people may not know that they have ADHD, and aren't
20 treated with conventional therapeutics, such as methylphenidate, and they don't know that their symptoms are associated with a known neurobehavioral disorder. If such people happen to try cocaine, they might find that it initially makes them more focused and able to deal with life, so they keep taking it, and they may become addicted.

25 Identification of genetic associations and correlations with the occurrence and severity of addiction and other behavioral disorders is not currently available in combination with genetic counseling for prevention, transmission and effective treatment. We have developed a program for providing a comprehensive

resource for physicians and their patients for the diagnosis and management of neurobehavioral disorders in a format that can be utilized easily and safely by the general practitioner. This resource can provide the physician to with proprietary genetic test and full evaluation of the patient's genetic profile, and to specific therapeutic treatments indicated for the patient. The evaluation can also advise the physician and family of nutritional and other non-pharmaceutical interventions which are specific for the patient's genetic profile. The testing and evaluation results can be available remotely through the Internet. Testing can be done in the physician's office with the data transmitted over the Internet for results and evaluation. In-home testing by the individual can be done with the results returned by mail and remotely over the Internet if confidentiality needs to be assured. Testing can be done on newborns, and screening of school children can be done to determine the level of risk and identify individuals who may benefit from preventative treatment.

Many of the above-described neurobehavioral disorders have a basis in genetics. For example, Table I below presents some previously reported associations between central neurobehavioral disorders and neurotransmitter, receptor and/or transporter genes.

20

Table I

Disorder	Neurotransmitter-Receptor/Transporter
Addictive (alcoholism, drug addiction)	<p>Adrenergic genes: ADRA2A, ADRA2C, DBH</p> <p>Catecholamine metabolism genes: monoamine oxidase alleles (MAOA), catechol-O-methyl transferase (COMT)</p> <p>Dopamine genes: subtypes D1, D2, D3, D4r and-D5, dopamine transporter gene (DAT1)</p> <p>GABA genes: GABAA3, GABAA6, GABAB3</p> <p>Serotonin genes: 5HT1A, 5HT1DB, 5HT2A, 5HT2C, 5HT3, TDO2</p>

	Others: alcohol dehydrogenase, cannabinoid, corticotrophin releasing factor, cholinergic (nicotinic) -- ALDH2, ADH2, ADH3, CNR1, CRF, CHRNA4, NMDAR1, PENk
ADD/ADHD	Adrenergic genes: ADRA2A, ADRA2C, DBH Catecholamine metabolism genes: MAOA alleles, COMT Dopamine genes: DRD1, DRD2, DRD5, DAT1 GABA genes: GABA genes: GABAA3, GABAB3 Serotonin genes: 5HT1A, TDO2 Others: CNR1, CHRNA4, NMDAR1, PENk
Anxiety	Adrenergic genes: ADRA2A, ADRA2C, DBH Catecholamine metabolism genes: MAOA alleles, COMT Dopamine genes: subtypes D1, D2, D3, D4 and D5, dopamine transporter gene (DAT1) GABA genes: GABAA3, GABAB3 Serotonin genes: 5HT1A, 5HT1DB, 5HT2A, 5HT2C, TDO2 Others: CNR1, CHRNA4, CRF, NMDAR1, PENk
Post-traumatic stress	DRD2, DAT1, mNOSIA

5 II. Intravenous Treatment of Neurobehavioral Disorders

According to certain embodiments of this invention, intravenous administration is provided of compositions designed to restore more normal neuronal and physiological function. In general, the substances that can be useful when injected intravenously for treating neurobehavioral disorders include amino acids, corticosteroids, vitamin C, gamma-globulin, compounds that inhibit neuropeptidyl opioid degradation, neurotransmitter precursors including amino acids, substances that promote neurotransmitter synthesis, substances that increase uptake and utilization of amino acids, substances that promote normal carbohydrate metabolism, dopamine receptor agonists, antiinflammatory agents, opioid receptor antagonists, substances that inhibit seizures, ammonia

scavengers, other immune enhancers and other substances described herein below.

It can be appreciated that, depending upon the neurobehavioral disorder to be treated, not all of the above substances need be administered together.

5 Rather, the compositions can be designed to affect those symptoms experienced by a particular patient having a neurobehavioral disorder. In general, substances that can be useful for the compositions of this invention can comprise one or more of the following substances:

(a) an amount of an endorphinase or enkephalinase inhibitor sufficient to
10 inhibit degradation of neuropeptidyl opioids selected from a group consisting of amino acids, peptides, and structural analogues or derivatives thereof including D-phenylalanine;

(b) a neurotransmitter synthesis-promoting amount of at least one neurotransmitter precursor selected from the group comprising dopamine
15 precursors L-tyrosine, L-phenylalanine, L-DOPA or from the group comprising serotonin precursors L-tryptophan, 5-hydroxytryptophan, or from the group comprising gamma amino butyric acid (GABA) precursors L-glutamine, L-glutamic acid, and L-glutamate;

(c) A tryptophan concentration enhancing amount of chromium picolinate or
20 chromium nicotinate or chromium polynicotinate;

Carbohydrate utilization can be increased by substances including chromium picolinate and chromium nicotinate. In certain embodiments, it can be desirable to use both because some genetically disposed individuals may not be able to utilize one or the other. Using both can help assure that the chromium
25 will be active and functional within the body.

(d) a chromium utilization-enhancing amount of vitamin B-6, including pyridoxal-5'-phosphate ("p-5-p");

- Pyridoxal-5-p can be useful to compensate for an inability of certain subjects to utilize vitamin B-6 in order to metabolize chromium. Pyridoxal-5'-p can promote prolonged recovery after treatment. Use of p-5'-p in this composition can be useful when used in conjunction with (a) above or any other compositions of this invention. Without sufficient chromium, carbohydrate metabolism can be impaired and carbohydrate craving, obesity, food sensitivity can occur. Chromium can also help to prevent diabetes and to assist in detoxification from chemical addiction. Moreover, improper pH in the gut can result in chromium loss or poor uptake, and can result in carbohydrate craving.
- 10 The continued consumption of carbohydrates without sufficient p-5'-p to support chromium utilization can alter the pH even further. In certain embodiments, it is not necessary to administer p-5'-p with exogenous chromium, because p-5'-p can aid the body in being able to use the chromium it has stored or taken in through food and water;
- 15 (e) an amino acid absorption and utilization enhancing amount of alpha-keto-glutarate;

Alpha-keto-glutarate can boost the synthesis of neurotransmitters made from amino acid precursors. Alpha-keto-glutarate can enhance the absorption and utilization of (a) and (b) and any of the amino acids comprising this composition;

20

- (f) an amount of oxytocin sufficient to increase hormonal action;
- (g) an amount of an opiate antagonist sufficient to inhibit the effects of an opiate at the receptor sites, including naloxone, which may be administered intravenously or taken orally after the intravenous treatment;

25 One theory that may account for the beneficial effects of naloxone and other opiate antagonists is that the antagonist may reduce an over abundance of receptors that may have been created by drug or heroin use, and thereby assisting

in reducing craving. Possibly the opioid receptor antagonist will effectively compete for the receptor sites, thereby reducing craving;

(h) an amount of a dopamine receptor agonist sufficient to activate post-synaptic dopamine receptors in the striatum, wherein said dopamine receptor agonist is bromocryptine or aprocosate or ondansetron.

(i) an amount of magnesium taurate sufficient to enhance the absorption of amino acids and the production of neurotransmitters from the amino acids and/or to restore magnesium levels;

In certain persons utilizing alcohol and some drugs, magnesium may become lost. Increasing magnesium can increase the efficacy of compositions comprising (a), (b) or (c) above, used alone or together, and other substances that comprise the composition. Magnesium taurate is an amino acid mineral complex that can promote bioavailability of the essential mineral magnesium;

(j) at least one amino acid or derivative thereof selected from essential and non-essential amino acids: L-aurine, L-tyrosine, L-phenylalanine, L-glutamine, D-phenylalanine, L-tryptophan, 5 hydroxytryptophan, arginine, methionine, leucine, isoleucine, histidine, valine, glycine, serine, asparagine, aspartic acid, citrulline, glutamic acid, ornithine and homocysteine at a dose of between about 1 µg and about 100 mg each, and if desired, an amount of citric acid sufficient to prevent adverse effects of ammonia generated by the infusion of amino acids. An example of a suitable mixed amino acid solution is Aminosyn™.

The amino acids can be advantageously infused intravenously over a short period of time (several minutes to several hours). However, infusion of a bolus of amino acids over such periods of time can result in the production of ammonia, which in sufficiently high concentrations can be neurotoxic. Therefore, in certain embodiments of compositions of this invention, citric acid can be added to reduce the levels of ammonia. Citric acid can spare glutamic acid and aspartic acid, each of which can aid in the removal of excess ammonia. Furthermore, an

alcoholic can have higher levels of ammonia in the brain due to use of alcohol to begin with, which would put him at risk of ammonia neurotoxicity. Thus, citric acid can be beneficial in treating people with alcohol addiction, even in the absence of exogenously administered amino acids;

- 5 (k) A brain seizure inhibiting amount of L-aurine or L-GABA.
(l) an ammonia reducing amount of a compound selected from the group consisting of alpha-keto-glutarate, magnesium, aspartic and glutamic acid.

High levels of ammonia, brain trauma, neurotoxic substances and many other conditions can result in brain seizures that may be responsible for the addict's need to self-medicate with drugs or alcohol, possibly carbohydrates and high-risk activities. Seizure activity or high rate of blood flow through certain areas of the brain, especially the cingulate gyrus area, can cause behavioral problems such as anxiety, obsessive thinking, obsessive compulsive disorder, anger and violent behavior disorders and other neurobehavioral disorders. In environmental sensitivity disorder, as well as other disorders, the patient can often be difficult and irritable and can lose the support of health professionals and family because of this condition. Also, taurine in sufficient amounts can increase assimilation of vitamin B-6, which can in turn promote uptake and utilization of chromium. The following will particularly aid in the reduction of ammonia:
10
15
20 alpha-keto-glutarate, magnesium, aspartic and glutamic acid

These substances can improve the results achieved when using (a) alone or any other substance in this composition with (a) above. Without the appropriate levels of these substances the patient may not feel comfortable may seek substances or activities with which to self medicate and thereby increase the risk of becoming addicted to the substances or activities. Taurine and GABA do not cause addiction and can allow neurotransmitter function to normalize. If unwanted behavior continues, the composition can be supplemented by aminomethyl-cyclohexaneacetic acid (Neurontin™; Parke Davis), Depakote™
25

(Divalproex Sodium, a delayed release of valproic acid) or any similarly acting compound. These agents may be given intravenously or orally, and the dosage may be increased until anger, hostility, rage, rigidity in thinking, obsessive disorders, and other neurophysiologically associated behaviors are abated. These substances can be useful to promote patient comfort and ease in order to maintain their recovery to continue to receive effective medical treatment without interruption by episodes of uncontrolled behavior, anger or paranoia. The use of valproic acid can be desirable because it has been shown that over time the compound can reverse seizure disorders.

- 5
10 (m) an anti-inflammatory agent selected from the group consisting of corticosteroids, vitamin C and glutamate;

Anti-inflammatory agents can preserve the availability of amino acids which the liver requires to manufacture neurotransmitters and other necessary substances for proper neurological function. Many conditions cause inflammation, notably an inflamed gut from drinking, drug use, food sensitivity (leaky gut syndrome), bacteria, viruses, toxic exposures, mycoplasma infections, parasitic infections or others. Anti-inflammatory agents can be used to spare amino acids during this treatment. Corticosteroids including cortisol, hydrocortisone and others can be useful. Corticosteroids can be synthetic or from adrenocortical extracts ("ACE"). In particular, L-glutamate can be useful. One possible mechanism of L-glutamate's action may be that it can decrease perforations in the lining of the gut which have been caused by alcohol or drug use, bacteria, viruses, food sensitivity, improper pH of the gut and by toxic substances. Additionally, vitamin C given intravenously can have anti-bacterial, anti-viral, anti-inflammatory effects.

- 25
(n) An immune system enhancing amount of gamma globulin.

Because of the distinct possibility of an untreated viral or bacterial infection in the patient, the immunological boost of gamma globulin is an active

compound in this composition. Dosages in the range of about 5-10 grams can be administered either alone or with other compounds, given over a four-hour period of time.

(o) a vitamin selected from the group consisting of niacin, niacinamide (including niacinamide ascorbate), vitamin B-complex, vitamin B-12, riboflavin (including riboflavin-5-phosphate), Ginko Biloba, N-acetylcysteine (glutathione), MSM, folic acid (including folacin), vitamin E, biotin, vitamin A, pantothenic acid (including calcium D-pantothenate), coenzyme Q10 for ammonia reduction and brain function, thiamin (including thiamin mononitrate and thiamin HCl), vitamin C including niacinamide ascorbate, vitamin A including beta-carotene, folic acid (including folacin), bioflavinoids, para-amino benzoic acid, pyridoxine HCl;

(p) an amount of a substance to improve the action of insulin selected from the group consisting of: iron (including iron aspartate/chelate), magnesium oxide, zinc chelate, selenium and vanadium, inositol, thiamin (including thiamin mononitrate and thiamin HCl), vitamin C including niacinamide ascorbate, vitamin A including beta-carotene, folic acid (including folacin), bioflavinoids, para-amino benzoic acid, pyridoxine HCl; and

(q) an amount sufficient to promote amino acid metabolism of a mineral, essential element or electrolyte selected from the group consisting of zinc (including zinc aspartate/picolinate/chelate), copper, chromium, manganese, selenium, potassium, calcium (including calcium chelate), sodium, and magnesium, including magnesium oxide/chelate.

III. Oral Compositions for Treatment

In certain embodiments of this invention, oral compositions can comprise all of the above-listed compounds. In other embodiments, compositions can include substances formulated as oral nutritional supplements and herbal

remedies. These oral nutritional supplements and herbal remedies can be administered either at the same time as the intravenous treatment, or thereafter. Oral compositions can include the above intravenous compositions listed in (a) - (q) above, and also can include:

- 5 (r) an amount of one or more substances to restore proper assimilation of nutrients in the body selected from the group consisting of betaine hydrochloride, gastric acid, Ox Bile, pancreatic enzymes sufficient to promote digestion, plant enzymes, fructo-oligosaccharides, normal bacterial flora including lactobacillus and/or bifidobaccillus, especially during or after the use of antibiotics, grapefruit
10 seed extract, grape leaf extract and/or oil of oregano, and black walnut hull extract;

Lactobacillus and bifidobacillus can be especially useful to restore normal flora after antibiotic treatments, and grapefruit seed extract can reduce growth of Candida albicans, and thereby can reduce neurotoxic substances produced by C.
15 albicans. Grape leaf extract and oil of oregano can act as anti-viral and anti-bacterial agents, and black walnut hull extract can act as an anti-parasitic agent

- (s) a blood pressure stabilizing amount of licorice root extract;

Licorice root extract can maintain blood pressure during treatment with Vitamin C given intravenously.

- 20 (t) A progesterone enhancing amount of wild Mexican yam;

Wild Mexican yam can be used orally by females over 20 to fortify insufficient amounts of progesterone in modern day foods.

- (u) An energy enhancing amount of Gensing extract; and

Ginseng extract will be used orally for men to support energy during
25 treatment.

- (v) an amount of a sulfation pathway enhancing compound selected from the group consisting of cysteine, taurine and salts of sulfate.

The liver can provide sulfation pathways for detoxifying drugs, steroid hormones and toxins. By increasing the availability of sulfation precursors, detoxifying can occur more rapidly.

5 In certain other embodiments, orally administered compositions can comprise the substances included above for the intravenous treatment, and can also include: Ginkgo Biloba, phosphatidylserine, phosphatidylcholine, alpha lipoic acid, red ginseng root, L-aspartic acid, ephedrine (also known as Ma Huang), pancreatic enzymes, plant-source enzymes, caffeine, (including that
10 found in Guarana or Coca), methyl-sulfonyl-methane ("MSM"), theobromine (including that found in chocolate), Hypericum perforatum, S-adenosyl methionine ("SAME"), dihydroxyacetate ("DHA"), DMAE, grape seed extract, betaine, prickly pear cactus, Gymnema sylvestre extract, nicotinamide adenine dinucleotide/hydrogen (NADH; coenzyme 1), cholecystokinin, Cyclo (His-Pro),
15 corticotropin-releasing hormone ("CRH"), neuropeptide Y, galanin, mono laurin and the substances in the following four compositions, which are, among others preferred for treatment for certain individuals.

 Some of the compositions used for intravenous infusion can be used orally after intravenous treatment in order to reduce the inflammation long-term
20 and also to help to heal the cause of inflammation. Vitamin C can be given orally at the same time as or after the intravenous infusion. One useful oral form of vitamin C is Ester-C™, used in a dose of about 5-20 grams daily, and a useful intravenous form of vitamin C is ascorbic acid. If administered separately, the dosage of intravenous vitamin C can be in the range of about 30-50 grams
25 depending on body size, in 500-ml saline solution over a four-hour period. The addition of minerals listed below may be added to this solution. A non-steroidal anti-inflammatory may be used intravenously or orally to temporarily reduce inflammation during treatment.

In addition to the compositions above administered intravenously and/or orally, patients physically capable of sustained exercise can benefit from exercise sufficient to cause release of amino acid stores from muscle tissue into plasma. For example, for many patients, 30 minutes on an exercise bicycle can be beneficial. Additionally, sleep medication or melatonin can be provided if the patient does not sleep well, so that the cortisol rise during sleep will occur correctly so that the liver will rebuild the muscle tissue from plasma during rest.

Table II below provides conversion factors for the formulations presented herein.

Table II
Conversion Factors for Formulas

<u>Substance</u>	<u>Volume Provided</u>	<u>Equivalent</u>
<u>Amount</u>		
Ca gluconate	10 cc	980 mg
Multi-Trace Elements		
Cr		4 µg
Cu		0.4 mg
Mn		0.1 mg
Zn		1 mg
Se		40 µg
Abram's Zn	1 cc	5 mg
American Regent		
Se	1 cc	40 µg
Cr	1 cc	4 µg
Mn	1 cc	0.1 mg
Cu	1 cc	0.4 mg
Abram's vitamin B-12	2 cc	1 mg
College vitamin B-6	1 cc	100 mg

Table III below provides sources of certain substituents of the compositions of this invention

Table III
Sources of Substituents

5	<u>Substituent</u>	<u>Source</u>
	Normal saline (0.45%)	Abbott Pharmacy
	Normal saline (0.45%)	Abrams Pharmacy
	Aminosyn™ amino acids	Abbot Pharmacy
10	Sodium Ascorbate	Merit Pharmaceuticals
	Taurine	College Pharmacy, Colorado Springs
	Vitamin B-1	Abrams Pharmacy, College Pharmacy
15	Vitamin B-5	Abrams Pharmacy, College Pharmacy
	Vitamin B-6	Abrams Pharmacy, College Pharmacy
	Vitamin B-12	Abrams Pharmacy
20	Vitamin B complex	College Pharmacy
	Adrenocortical extract	Rocky Mountain Pharmacy, Phoenix AZ
	Vitamin A	Abram's Pharmacy, Dallas TX
	Vitamin E	Abrams Pharmacy
25	Multi-Trace Elements	Merit

Example 1: Compositions for Intravenous Infusion I

30	To a solution of balanced amino acids such as "Aminocyn™," add:	
	L-aurine	about 5 grams
	L-tyrosine	about 5 grams
	L-glutamine	about 8-10 grams
	D-phenylalanine	about 5 grams
	L-phenylalanine	about 5 grams
35	5-hydroxytryptophan	about 2 grams
	or tryptophan	about 2 grams
	L-glutathione	about 3 grams
	methionine	about 1 gram

32

	magnesium oxide	about 1 gram
	calcium	about 1 gram
	zinc	about 200 mg
5	vitamin B-Complex	about 200 mg
	vitamin B-12	about 200 mg
	p-5'-p	about 200 mg
	potassium	about 100 mg
	alpha-keto-glutarate	about 200 to about 600 mg
	chromium nicotinate	about 20 mg to about 5 grams
10	chromium picolinate	about 20 mg to about 5 grams
	copper	about 10 mg
	manganese sulfate	about 1 mg
	citric acid, aspartic acid and/or glutamic acid	about 200 mg to about 6
15	grams	

Example 2: Compositions for Intravenous Infusion II

To a solution of balanced amino acids such as "Aminocyn™," add:

20 about 30 - about 50 mg ascorbic acid or calcium ascorbate in 500 ml
normal saline.

Alternatively, add any of the substances from Example I above.

25

Example 3: Compositions for Intravenous Infusion III

To a solution of balanced amino acids such as "Aminocyn™," add:

30 Gamma-globulin.

Alternatively, add any of the substances from Example I above.

The method of infusing the substances intravenously works rapidly because of direct access by the intravenously infused substances to the brain. It can rapidly reduce symptoms and can begin to change behaviors. The methods of this invention can reduce or eliminate craving for addictive substances during the initial treatments and over the long term. Treatment can be repeated if craving returns. This method may be followed with oral administration of any of

35

the compounds included above that contribute to recovery.

This method may be used to treat any of the neurobehavioral disorders and can be particularly effective in treating addiction disorders, obesity, compulsive and anxiety disorders; mood disorders and other disorders that can result in acts of violence. The compositions and methods of this invention in
5 combination with medications for seizures and seizure induced behavioral or attentional disorders can also be effective.

The following three Formulas can be used to treat addiction to drugs, alcohol and other disorders. The drug of choice can be an indicator of which
10 formula(s) can be useful to treat patients suffering from addiction.

Formula I can be advantageously used to treat patients addicted to cocaine, caffeine, tobacco, marijuana, sugar, aspartame, and amphetamines. Formula I can also be desirably used to treat patients suffering from ADHD or other attentional disorders. To treat patients suffering from compulsion to
15 gamble, or compulsion to high risk-taking behavior or addiction to sex, Formulas I and/or II described herein can be desirable.

Formula II can be advantageously used to treat patients addicted to alcohol, carbohydrates, heroin, morphine and codeine. To treat patients experiencing high anxiety or paranoia, it can be desirable to use Formula II
20 followed by Formula III described herein. For patients experiencing obsessive-compulsive disorder, panic disorder, disorders of violence and/or rage, it can be desirable to use Formula II.

Formula III can be desirable to treat patients addicted to carbohydrates, when combinations of drugs are the drugs of choice, including, but not limited to
25 combinations of alcohol, cocaine and alcohol. Formula III can also be advantageously used to treat patients who are incoherent, will not disclose the drug(s) of choice, or where an emergency situation is present. Formula III can also be desirably used to treat severely depressed patients.

Example 4:**Formula I: Formula for Patients Needing Neurological Stimulation**

5	Substance	Preferred Amount	Range
	L-tyrosine	5000 mg	4000 - 7000 mg
	L-glutamine	1000 mg	2500 - 3000 mg
	L-tryptophan	1000 mg	1000 - 5000 mg
	(or 5HTP)	1000	1000 - 5000 mg
10	L Phenylalanine	4000 mg	3000 - 6000 mg
	D-phenylalanine	5,500	3000 - 7000 mg
	L-glutathione	1000 mg	250 - 3000 mg
	Alpha-keto glutarate	200 mg	50 - 400 mg
	P-5-p	200 mg	50 - 400 mg
15	Thymus extract	4 ml	2 - 4 ml
	Lidocaine (1%)	10 mg	5 - 10 mg
	Na ascorbate	25 gm	5 - 50 gm
	Cr polynicotinate	12 µg	8 - 16 µg
20	nicotinate or picolynate		
	Zn chelate	360 mg	200 - 1500 mg
	MgO or Mg taurate	1000 mg	500 - 1500 mg
	Ca chelate	1000 mg	500 - 1500 mg
	vitamin B12	1.5 mg	0.5 - 3 mg
25	vitamin B12 push	1 mg	0.5 - 1.5 mg
	vitamin B complex	1 mg	0.5 - 1.5 mg
	vitamin B5	100 mg	50 - 150 mg
	vitamin B6	100 mg	50 - 150 mg
	vitamin B1	100 mg	50 - 150 mg
30	folic acid	0.066 mg	0.03 - 0.09
	oxytocin	2 ml	1 - 10 ml
	Balanced amino acid solution with electrolytes,	1000 ml	

Example 5:**Formula II: For Patients Needing Neurological Calming**

	Component	Preferred amount	Range
5	L-aurine	1000 mg	150 - 2000 mg
	L-tyrosine	1500 mg	500 - 1500 mg
	L-glutamine	8000 mg	7000 - 10,000 mg
	L-tryptophan	3600 mg	1000 - 4000 mg
10	(or 5HTP)	3600	3000 - 7000 mg
	L Phenylalanine	3000 mg	1000 - 6000 mg
	D-phenylalanine	5,500	1000 - 7000 mg
	L-glutathione	1000 mg	250 - 3000 mg
	Alpha-keto glutarate	200 mg	50 - 400 mg
15	P-5-p	200 mg	50 - 400 mg
	Thymus extract	4 ml	2 - 4 ml
	Lidocaine (1%)	10 mg	5 - 10 mg
	Na ascorbate	20 gm	10 - 60 gm
	Cr polynicotinate	12 µg	8 - 16 µg
20	Zn chelate	360 mg	200 - 1500 mg
	MgO or Mg taurate	1000 mg	500 - 1500 mg
	Ca chelate	1500 mg	1200 - 2500 mg
	vitamin B12	1.5 mg	0.5 - 3 mg
	vitamin B12 push	1 mg	0.5 - 1.5 mg
25	vitamin B complex	1 mg	0.5 - 1.5 mg
	vitamin B5	100 mg	50 - 150 mg
	vitamin B6	100 mg	50 - 150 mg
	vitamin B1	100 mg	50 - 150 mg
	folic acid	0.066 mg	0.03 - 0.09
30	oxytocin	2 ml	1 - 10 ml
	Balanced amino acid solution with electrolytes,	1000 ml	

Example 6:**Formula III: A Universal infusion when the drug of choice is not known**

	Component	Preferred Amount	Range
5	L-aurine	5000 mg	150 - 7000 mg
	L-tyrosine	5000 mg	4000 - 7000 mg
	L-glutamine	8000 mg	7000 - 10,000 mg
	L-tryptophan	2000 mg	1000 - 4000 mg
	(or 5HTP)	5000	3000 - 7000 mg
10	L Phenylalanine	4000 mg	3000 - 6000 mg
	D-phenylalanine	5,500	3000 - 7000 mg
	L-glutathione	1000 mg	250 - 3000 mg
	Alpha-keto glutarate	200 mg	50 - 400 mg
	P-5-p	200 mg	50 - 400 mg
15	Thymus extract	4 ml	2 - 4 ml
	Lidocaine (1%)	10 mg	5 - 10 mg
	Na ascorbate	20 gm	10 - 60 gm
	Cr polynicotinate	12 µg	8 - 16 µg
	Zn chelate	360 mg	200 - 1500 mg
20	MgO or Mg taurate	1000 mg	500 - 1500 mg
	Ca chelate	1000 mg	500 - 1500 mg
	vitamin B12	1.5 mg	0.5 - 3 mg
	vitamin B12 push	1 mg	0.5 - 1.5 mg
	vitamin B complex	1 mg	0.5 - 1.5 mg
25	vitamin B5	100 mg	50 - 150 mg
	vitamin B6	100 mg	50 - 150 mg
	vitamin B1	100 mg	50 - 150 mg
	folic acid	0.066 mg	0.03 - 0.09
	oxytocin	2 ml	1 - 10 ml
30	L-leucine	450 mg	300 - 1500 mg
	Balanced amino acid solution with electrolytes,	1000 ml	

Example 7: Intravenous Ethanol Treatment I

- 35 To carry out a method according to these embodiments of the invention, an ethanol solution, preferably about 10 % to about 20% by volume, is intravenously infused into the alcohol dependent individual. The concentration of alcohol in solution can vary from 5% to 25% by volume. Ethanol concentrations

of substantially less than 5% have been found less effective in treatment and concentrations in excess of 25% can cause discomfort at the administration site. One suitable alcohol solution for infusion which is available in prepackaged form is manufactured by Kendall-McGaw Laboratories, Inc., and contains 5%
5 dextrose and 10% ethanol in distilled water. This solution is intended for nutrition, and therefore includes dextrose. Although there is actually no need for dextrose when treating alcohol dependence, its presence does not create any complications or adverse effects.

The infusion procedure is performed according to standard techniques
10 and procedures. In brief, an infusion bag or bottle containing the solution is hung from a support. A drip connector is attached to the bottle, and tubing is attached to the drip connector. Air is then expelled from the tubing, and after clamping the tubing, a needle is attached to the tubing. The vein is then punctured with the needle, and the needle is immobilized against the arm. The drip connector allows
15 adjustment of the infusion rate.

A typical administration program, using the 5% dextrose and 10% ethanol solution, begins with infusion of 220 ml of solution twice daily on the first day. At least about two and one-half hours rest or waiting period is provided between successive infusions.

20 For most patients, it can be desirable to infuse less than about 220 ml in one hour. The metabolic rate of ethanol is often not more than 20 ml per hour, and if 220 ml of a 10% ethanol solution is infused in one hour, then y more alcohol is being infused than may be metabolized. However, the rate of infusion can vary greatly, and can exceed these limits, depending on the weight of the
25 patient and especially on his tolerance for alcohol.

Once the infusion is progressing, the rate of infusion can be gradually increased if the patient appears tolerant. The patient is monitored throughout the infusion for adverse reactions. If these appear, the infusion rate is decreased.

Example 8: Intravenous Ethanol Treatment II

Following the above-described treatment on the first day, a treatment schedule, using the aforementioned 10% ethanol solution, is as follows:

- 5 Day two: 200 ml, twice daily
- Day three: 180 ml, twice daily
- Day four: 160 ml, once daily
- Day five: 140 ml, once daily
- Day six: 120 ml, once daily
- Day seven: 100 ml, once daily
- 10 Day eight: 80 ml, once daily
- Day nine: 60 ml, once daily
- Day ten: 40 ml, once daily.

For the second and third days on which two infusions are given, at least
15 two and one-half hours can desirably be provided between successive infusions. The recommended infusion rate can be determined using the guidelines and considerations mentioned above. In general, it can be desirable to limit the rate of infusion to less than about 20 ml per hour. However, the infusion rate can exceed this limit depending on patient's weight and tolerance.

20 As noted above, solutions with different concentrations of alcohol, i.e. from about 5 to 25% can be used in treatment. When the concentration is varied, the treatment schedule, and the guidelines for calculating the infusion rate need not change. The volume of solution is adjusted so that the same volume of alcohol as set forth above is infused with each treatment. For example, if using a
25 20% ethanol solution, one-half the solution volume set forth above is infused each time. Thus, 10 ml of the 20% solution is infused twice on the first day, 100 ml is infused twice on the second day, and so on through the tenth day, at which time 20 ml is infused once.

Although an initial ten-day treatment program is preferred and has been
30 found effective in most patients, a shorter or longer program can also be used. In fact, for those with severe alcohol dependence, a longer treatment program, e.g.,

up to twenty days, is recommended. Further, a greater or lesser quantity of alcohol than listed above can be infused, and a greater or lesser number of treatments can be administered each day.

Recidivism can often be effectively re-treated. The re-treatment program is generally of shorter duration, and preferably lasts for six days. With the shorter treatment the initial quantity of alcohol infused is preferably the same as with the ten day treatment, i.e., on the first day preferably about 40 to 50 ml of ethanol is administered.

Example 9: Intravenous Ethanol Treatment III

In cases of recidivism, it can be desirable to use a 10% ethanol solution and to administer a dose of 22 ml twice on the first day. The amount of solution infused is then reduced as follows:

Day two: 200 ml, twice daily
Day three: 180 ml, twice daily,
Day four- 160 ml, once daily,
Day five: 140 ml, once daily
Day six: 120 ml, once daily.

If the shorter treatment program is not effective, a longer re-treatment program can be used. Further, the volume of solution, the rate of infusion, and the ethanol concentration can all be varied with the shorter program in the same manner, and subject to the same considerations, in which they are varied in the longer ten-day program.

In patients with drug addiction, prescription drug addiction, nicotine, carbohydrate or other substance addiction, either the ethanol is used in the IV infusion (when the addicts drug of choice mimics the dopamine response that occurs when ethanol is consumed); or the active substance in the drug of choice is used in the IV infusion in decreasing amounts consistent with the process stated above.

Example 10: Case Study of a Patient with Neurobehavioral Disorders I

5 A patient, white male, age 38, weight 180 lbs presented with sleep disorders, obsessive-compulsive disorder, depression, anger and rage disorder, drug and alcohol addiction, attention deficit hyperactivity disorder, environmental illness, neurally mediated hypotension, chronic fatigue syndrome and dyslexia. The subject had a history of debilitating brain injury and by about 11 year of age, 10 was unable to arise in the morning. The subject became allergic to foods and chemicals by age 15. By age 25, the subject was unable to attend public school, complete home study or do outside work consistently. The subject is athletic and highly intelligent, but suffered from obsessive ideation, worrying, inability to cope with change, inability to eliminate negative thoughts, obsessive compulsive 15 behaviors, a high level of irritability, argumentative interpersonal relations and cognitive inflexibility. The subject also experienced short attention span, distractability, impulse control problems, violent outbursts, social anxiety and poor judgement.

 Studies of brain perfusion by SPECT methods showed increased 20 perfusion in the cingulate gyrus, decreased perfusion in the pre-frontal cortex, temporal lobes limbic system and basal ganglia.

 Conventional therapy including antidepressive and anti-hypotensive agents were minimally effective at restoring the subject's neurobehavioral state.

 Therapy was begun with an intravenous infusion I of the following 25 composition:

Infusion 1:

1000 ml	normal saline
30 grams	sodium ascorbate
30 10 grams	ascorbic acid

41

4.5 mg calcium gluconate
10 cc Mg
0.11 mg Mn
4 cc pantothenic acid
5 1 cc vitamin B-6
1 mg zinc; and
40 µg selenium.

10 The subject showed no significant response.

Infusion 2:

15 5 grams of gamma globulin (Gammagard) administered 4 times.

The subject experienced mild response. An administration of 10 grams improved the subject's state of fatigue.

Infusion 3:

20 500 ml. normal saline
45 - 50 grams Na ascorbate
250 µg Mo
300 mg Mg
1 cc Mn
25 3 cc Multi pack of 5 minerals (Cr, Zn, Se, Cu, Mn)
1 cc Zn
2 cc selenium
2 cc Cr
5 millieq KCl
30 3 cc Ca gluconate
1 cc lidocaine

The subject noted some improvement in muscle and joint discomfort.
Decreased anxiety and paranoia. Subject felt relaxed.

35 **Infusion 4:**

500 ml normal saline
45-50 grams Na ascorbate

42

	250 mg	Mo
	600 mg	Mg
	1 cc	Mn
	3 cc	Multi pack of 5 minerals (as in Infusion 3)
5	1 cc	Zn
	2 cc	selenium
	2 cc	Cr
	5 millieq	KCl
	3 cc	Ca gluconate
10	1 cc	lidocaine

The subject noted increased sense of comfort and well being by doubling the amount of Mg and continuing the lidocaine. Overall body pain, muscle pain and joint pain were reduced. Less reactive to allergens.

15 **Infusion 5:**

	500 ml	normal saline
	250 mg	Mo
	600 mg	Mg
	1 cc	Mn
20	3 cc	Multi pack of 5 minerals (as in Infusion 3)
	1 cc	Zn
	2 cc	selenium
	2 cc	Cr
	5 millieq	KCl
25	3 cc	Ca gluconate
	1 cc	lidocaine
	5 cc	taurine

Subject noted significant improvement in symptoms, less frantic, more stable, able to sleep better, positive mood enhancement and significant calming. Does not feel as overwhelmed.

Infusion 6:

	500 ml	normal saline
35	45-50 mg	Na ascorbate
	250 mg	Mo

43

- | | | |
|----|--|---|
| | 750 mg | Mg |
| | 1 cc | Mn |
| | 3 cc | Multi pack of 5 minerals (as in Infusion 3) |
| | 1 cc | Zn |
| 5 | 2 cc | selenium |
| | 2 cc | Cr |
| | 5 millieq | KCl |
| | 3 cc | Ca gluconate |
| | 5 cc | taurine |
| 10 | The subject noted further improvement of symptoms. | |

Infusion 7:

Repeat of Infusion 6. Subject noted similar symptoms as after Infusion 6.

15 Infusion 8:

- | | | |
|----|-----------|---|
| | 500 ml | normal saline |
| | 45-50 mg | Na ascorbate |
| | 500 mg | Mo |
| | 900 mg | Mg |
| 20 | 1 cc | Mn |
| | 3 cc | Multi pack of 5 minerals (as in Infusion 3) |
| | 1 cc | Zn |
| | 2 cc | selenium |
| | 2 cc | Cr |
| 25 | 5 millieq | KCl |
| | 3 cc | Ca gluconate |
| | 5 cc | taurine |

Subject noted improvement in symptoms.

30 Infusion 9:

- | | | |
|----|----------|---|
| | 500 ml | normal saline |
| | 45-50 mg | Na ascorbate |
| | 250 mg | Mo |
| | 600 mg | Mg |
| 35 | 1 cc | Mn |
| | 3 cc | Multi pack of 5 minerals (as in Infusion 3) |

44

1 cc Zn
2 cc selenium
2 cc Cr
5 millieq KCl
5 3 cc Ca gluconate
1 cc taurine
1 cc Cu solution
5 cc Adrenal cortical extract

Subject noted improved sleep patterns associated with the reduced
10 taurine.

Infusion 10:

500 ml normal saline
60 mg Na ascorbate
15 250 mg Mo
600 mg Mg
1 cc Mn
3 cc Multi pack of 5 minerals (as in Infusion 3)
1 cc Zn
20 2 cc selenium
2 cc Cr
5 millieq KCl
7 cc Ca gluconate
1 cc taurine
25 1 cc Cu solution
5 cc Adrenal cortical extract
250 mg glutathione

Subject noted less sensitivity to chemicals and was able to maintain
30 mental focus.

Infusion 11:

500 ml normal saline
60 mg Na ascorbate
35 600 mg Mg
1 cc Zn

45

	2 cc	selenium
	7 cc	Ca gluconate
	1 cc	taurine
	1 cc	Cu solution
5	5 cc	Adrenal cortical extract
	100,000 IU	vitamin A

Subject noted increased ability to change visual field.

10 **Infusion 12:**

	500 ml	normal saline
	40 mg	Na ascorbate
	250 mg	Mo
	600 mg	Mg
15	1 cc	Mn
	3 cc	Multi pack of 5 minerals (as in Infusion 3)
	1 cc	Zn
	2 cc	selenium
	2 cc	Cr
20	5 millieq	KCl
	7 cc	Ca gluconate
	1 cc	taurine
	2 cc	Cu solution
	5 cc	Adrenal cortical extract
25	100,000 IU	vitamin A

Subject noted reduced desire for cocaine, marijuana and alcohol.

Infusion 13:

30	500 ml	normal saline
	25 mg	Na ascorbate
	250 mg	Mo
	600 mg	Mg
	500 IU	vitamin E
35	1 cc	vitamin B1 + B complex
	2 cc	Mn
	1 cc	Zn

46

	2 cc	selenium
	2 cc	Cr
	7 cc	Ca gluconate
	2 cc	taurine
5	2 cc	Cu solution
	5 cc	Adrenal cortical extract
	100,000 IU	vitamin A

Subject noted reduction in craving. Fluid retention improved and blood pressure stabilized. Increased sense of calming. Increased motivation, mood and energy.

Infusion 14:

	500 ml	normal saline
15	25 mg	Na ascorbate
	250 mg	Mo
	600 mg	Mg
	500 IU	vitamin E
	1 cc	vitamin B1 + B complex
20	2 cc	Mn
	1 cc	Zn
	2 cc	selenium
	2 cc	Cr
	7 cc	Ca gluconate
25	2 cc	taurine
	2 cc	Cu solution
	5 cc	Adrenal cortical extract
	100,000 IU	vitamin A
	1.5 cc	vitamin B-12
30	2 ml	thymus extract

Subject noted increased strength, less need for sleep, less susceptible to colds, substantial increase in energy.

35 Infusion 15:

	500 ml	normal saline
--	--------	---------------

47

	250 mg	Mo
	1500 mg	MgCl ₂
	500 IU	vitamin E
	1 cc	vitamin B1 + B complex
5	2 cc	Mn
	6 cc	Zn
	2 cc	selenium
	2 cc	Cr
	10 cc	Ca gluconate
10	7 meq	KCl
	2 cc	taurine
	2 cc	Cu solution
	5 cc	Adrenal cortical extract
	50,000 IU	vitamin A
15	2 cc	thymus extract
	1 cc	lidocaine
	1.5 cc	vitamin B12

Subject noted greater desire for exercise and improved endurance. All
 20 body pains gone. Happy thoughts appear. Improved sleep pattern.

Infusion 16:

	300 cc	3.5 % Aminocyn
	450 mg	MgCl ₂
25	10 cc	Ca gluconate
	5 meq	KCl
	1.5 cc	vitamin B-12
	3 ml	oxytocin

Subject reported 50% decrease in addictive substances, increased
 30 calmness and happiness. Eager to interact socially. Obsessive thoughts and
 anxiety decreased. Substantially increased muscle strength

Infusion 17:

600 cc 3.5 % Aminocyn

35 Subject reported no adverse effects.

Infusion 18:**Formula III:**

	Component	Amount
	L-aurine	5000 mg
5	L-tyrosine	5000 mg
	L-glutamine	8000 mg
	L-tryptophan	2000 mg
	(or 5HTP)	5000
	L Phenylalanine	4000 mg
10	D-phenylalanine	5,500
	L-glutathione	1000 mg
	Alpha-keto glutarate	200 mg
	P-5-p	200 mg
	Thymus extract	4 ml
15	Lidocaine (1%)	10 mg
	Na ascorbate	20 gm
	Cr polynicotinate	12 µg
	Zn chelate	360 mg
	Mg oxide	1000 mg
20	Ca chelate	1000 mg
	vitamin B12	1.5 mg
	vitamin B12 push	1 mg
	vitamin B complex	1 mg
	vitamin B5	100 mg
25	vitamin B6	100 mg
	vitamin B1	100 mg
	folic acid	0.066 mg
	Balanced amino acid solution with electrolytes,	1000 ml
30	Subject reported cravings eliminated. No withdrawal symptoms and no ideation of the addictive substance noted. Subject reported no adverse effects and was eager to leave the house. Subject appeared to be present, aware of surroundings. Judgement improved, improved self confidence.	

Infusion 19: Formula II without Thymus extract

	Component	Amount
	L-aurine	1000 mg
5	L-tyrosine	1500 mg
	L-glutamine	8000 mg
	L-tryptophan	3600 mg
	(or 5HTP)	3600
	L Phenylalanine	3000 mg
10	D-phenylalanine	5,500
	L-glutathione	1000 mg
	Alpha-keto glutarate	200 mg
	P-5-p	200 mg
	Thymus extract	4 ml
15	Lidocaine (1%)	10 mg
	Na ascorbate	20 gm
	Cr polynicotinate	12 µg
	Zn chelate	360 mg
	MgO	1000 mg
20	Ca chelate	1500 mg
	vitamin B12	1.5 mg
	vitamin B12 push	1 mg
	vitamin B complex	1 mg
	vitamin B5	100 mg
25	vitamin B6	100 mg
	vitamin B1	100 mg
	folic acid	0.066 mg
	2 ml	oxytocin
30	Balanced amino acid solution with electrolytes,	1000 ml

Subject noted craving eliminated. Sleep patterns improved. Noted that a change in life could be tolerated. No abdominal pain. Sense of doom eliminated.

Infusion 20: Formula II

35 Subject noted brighter mood than in infusion 19, with mood and energy levels improved.

Infusion 21: Formula I below without lidocaine and thymus extract

	Substance	Amount
	L-tyrosine	5000 mg
5	L-glutamine	1000 mg
	L-tryptophan	1000 mg
	(or 5HTP)	1000
	L Phenylalanine	4000 mg
	D-phenylalanine	5,500
10	L-glutathione	1000 mg
	Alpha-keto glutarate	200 mg
	P-5-p	200 mg
	Thymus extract	4 ml
	Lidocaine (1%)	10 mg
15	Na ascorbate	25 gm
	Cr polynicotinate	12 µg
	Zn chelate	360 mg
	Mg oxide	1000 mg
	Ca chelate	1000 mg
20	vitamin B12	1.5 mg
	vitamin B12 push	1 mg
	vitamin B complex	1 mg
	vitamin B5	100 mg
	vitamin B6	100 mg
25	vitamin B1	100 mg
	folic acid	0.066 mg
	2 ml	oxytocin
	Balanced amino acid solution with electrolytes,	1000 ml
30	Subject noted complete absence of craving, obsessive and/or negative thoughts. Feels very positive.	

Infusion 22: Formula I above for 7 consecutive days.

	Subject remained clear headed, jovial, beginning to plan for the future.	
35	Subject states that he feels "normal." Sense of humor returned, light hearted and quite confident. Subject appears to continue improvement.	

Infusion 23: Formula I, 6 months after infusion 22, for three consecutive days.

Subject reported feeling as good after one infusion as on the day of completion of infusion 22.

5 **Example 15: Case Study in a Patient with Neurobehavioral Disorders II**

 A 62 year old, white female, weight 140 lbs presented with a history of environmental sensitivity, food sensitivity. After a four-week hospitalization, she was advised that it was unlikely that she would work again because of her
10 immune over-reactivity to chemicals and foods. A diagnosis of chronic fatigue syndrome and infectious venulitis was made. The patient suffered from intermittent depression, anxiety disorder, panic disorder and attention deficit hyperactivity disorder and fibromyalgia. The subject suffered from severe craving and addiction to sugar, alcohol and caffeine.

15 The subject was unable to work for a period of eight years and lived in an isolated dwelling devoid of automobile traffic, hydrocarbons, gas fired heat or hot water, paints, synthetic materials, and cleaning agents. The subject's diet was one food at each meal, three times per day, on a strict rotation schedule. Only pesticide- and herbicide-free foods were used. Clothing was made of pesticide-
20 free natural fabrics. Only distilled water was provide. Conventional therapies were not successful.

 Therapy comprised a first intravenous infusion, over a three-hour period, of an intravenous formula consisting of:

Infusion 1:

25 400 ml normal saline
 20 gm ascorbic acid
 20 grams Na ascorbate
 10 cc Mg
 20 mg potassium

52

2 cc pantothenic acid
2 cc pyridoxine
1 cc selenium; and
10 cc calcium.

5

After treatment, the subject reported feeling more relaxed and less anxious.

Infusion 2: A second infusion, about 2 years later:

10 400 ml normal saline
 30 grams ascorbic acid
 30 grams Na ascorbate
 10 cc Mg
 20 mg potassium
15 2 cc pantothenic acid
 2 cc pyridoxine
 1 cc selenium
 10 cc calcium

The subject reported more relaxed and the depression completely abated.

20

Infusion 3: Repeat of the infusion 2.

Subject reported completely relieved of depression and anxiety. Subject reported that energy levels increased.

25 **Infusion 4:**

 400 ml normal saline
 25 grams ascorbic acid
 25 grams Na ascorbate
 1050 mg MgCl₂.

30 Subject noted marked increase in energy. Craving for sugar ceased. Subject felt optimistic and noted reduced adverse sensitivities to foods and to pollens and grasses.

Infusion 5: During influenza infection.

400 ml normal saline
25 grams ascorbic acid
25 grams Na ascorbate
5 1000 mg $MgCl_2$
750 mg calcium gluconate.

Subject's severe influenza symptoms ceased within 12 hours, fibromyalgia and muscle tenderness ceased. Subject noted increased self confidence.

10

Infusion 6: Same as infusion 5, but with the addition of 12,500 U vitamin A.

Subject noted no further change.

Infusion 7:

15 Subject was administered 10 grams Sandoglobulin intravenously every two weeks for a period of about 4 years. On alternating weeks, the subject was administered 40 - 50 grams vitamin C, 750 - 1250 mg $MgCl_2$, Ca gluconate, 500 - 750 mg (i.v.). Subject combined a non-steroidal anti-inflammatory agent, 200 mg one hour before administration of gamma globulin.

20 The subject experienced cessation of all food allergies and about 80 % reduction in chemical sensitivities. Subject noted about 95 % reduction in muscle tenderness and body and joint pain. Chronic inflammation of the neck ceased. Food allergies did not return for a period of 48 months, and then, only about 5% were experienced. Subject was able to resume general activity in environments
25 previously not tolerated. After the second infusion period, the subject resumed unlimited activities. The time to recovery after temporary exposure to chemical fumes was reduced from weeks or months to periods of less than one hour.

30

Infusion 7:

100 cc Aminocyn™
200 ml normal saline.

The subject tolerated the amino acid formula with no adverse affects.

5

Infusion 8:

400 ml normal saline
30 grams ascorbic acid
30 grams Na ascorbate
10 600 mg MgCl₂
2 cc glutathione
100 cc essential amino acids
2 cc pantothenic acid
2 cc pyridoxine
15 1 cc selenium
100,000 IU vitamin A
1000 mg Ca gluconate
8 µg Cr
20 10 mg Zn

Subject noted reduction in anxiety and a moderate sense of confidence and well being. Multi-tasking was reported to be easier, and an ability to make small decisions with fear of failure improved.

25 **Infusion 9:**

400 m. normal saline
600 mg MgCl₂
1 gram D-phenylalanine

Subject noted marked reduction in craving for sugars, caffeine and
30 alcohol.

Infusion 10:

400 ml normal saline

55

5 grams D-phenylalanine

Subject reported further reductions in cravings.

Infusion 11:

5 400 ml normal saline
 1 gram D-phenylalanine
 1 gram Chromium nicotinate

10 Subject reported having no cravings or ideation about addictive
 substances.

Infusion 12:

 400 ml normal saline
 600 mg MgCl₂
15 1 gram D-phenylalanine
 200 mg alpha-keto glutarate

Subject noted increase ability to focus and concentrate.

Infusion 13:

20 400 ml normal saline
 1 gram D-phenylalanine
 4 ml thymus extract

 Subject noted highly energized and still comfortable and without anxiety
 or cravings.

25

Infusion 14:

 400 ml normal saline
 1 gram D-phenylalanine
 10 mg lidocaine

30 Subject reported pain and achiness nearly gone. Subject engaged in
 exercise for the first time in 5 years.

Infusion 15:

Formula I above.

Subject felt sharper, more focused and energetic. Subject became able to organize business and personal matters. Sense of humor and relief increased.

5

Infusion 16:

Formula II above.

Subject noted difficulty arising after a night's rest, felt spacey.

10 **Infusion 17:**

Formula III

Subject noted results similar to those achieved after Formula I (infusion 15).

15 **Infusion 18:**

Formula I for 12 consecutive days.

Subject noted complete relief from depression and anxiety. Subject's energy reported to increase. Little or no body pain noted. Work output increased significantly. No thoughts of sugar or alcohol.

20

Infusion 19:

Subject foregoes conventional therapy for ADHD for 7 days and then had an infusion of Formula I.

Subject noted 80 % reduction in ADHD symptoms during a seven-day experimental period and has had no resumption of cravings.

25

The results of the two case studies demonstrate improvement in subjective and objective measures of neurobehavioral disorders.

IV. Other Methods of Delivering the Compositions

In addition to intravenous and oral administration, one can administer the compositions of this invention using the following routes: transdermal delivery using skin patches, intranasal spray, oral spray, eye drops, powders, pills, capsules, electrically stimulated delivery across the skin, as well as other methods of causing the substances to be transported across the skin, be absorbed into the body, inserted rectally, mouth sprays, drinks, bars, creams, foams, tooth pastes, covalently bonded to lipids for intramuscular injection, and also can be used for the fortifying of specialized soils to increase the amounts of these substances in foods natural foods.

In certain other embodiments of this invention, one can combine conventional pharmaceutical drug treatments along with the above-described methods. In certain embodiments, it can be desirable to provide conventional pharmaceutical treatments after administration of the intravenous treatment. In other embodiments, it can be desirable to provide pharmaceutical treatments while taking the oral supplements, and/or using a transdermal delivery system containing amino acid formulations. Several conventional and some new pharmaceutical medications can be desired. Naloxone can be used when the drug of choice is an opiate. Gamma vinyl-GABA can be useful in patients dependent upon nicotine.

Procaine hydrochloride or lidocaine, 1 % solution, may be used in patients having cocaine dependence. Procaine can neutralize effects of cocaine. However, it can be desirable to replace of neurochemicals depleted during cocaine dependence states.

VI. Diagnostic Methods for Diagnosing Neurobehavioral Disorders and Evaluating Therapeutic Efficacy

A. SPECT Scanning, Diagnosis and Treatment

Brain single photon emission computed tomography ("SPECT") imaging
5 is a method for studying cerebral perfusion and the effects of neuroactive
materials on perfusion in selected areas of the brain or generally over a wider
area. SPECT can be carried out using a device which utilizes minute doses of
radioactive isotopes bound to neurospecific pharmaceuticals and, indirectly, can
be used to analyze brain metabolic activity. It has been used for years in the
10 research of neurological and psychiatric disorders, along with positron emission
tomography ("PET") scanning to detect metabolic activity. SPECT is now
considered as accurate as PET and is less costly and subjects the patient to less
radiation.

SPECT findings can correlate with Attention Deficit Hyperactivity
15 Disorder ("ADHD"), Attention Deficit Disorder ("ADD"), brain trauma, bipolar
disorder, learning disabilities, temporal lobe dysrhythmias, addictive disorders
and disorders that result in violent behavior.

SPECT can detect cerebral perfusion abnormalities in substance abusers
in brain areas known to be involved in behavior, such as the frontal and temporal
20 lobes. Cocaine and methamphetamine are rapidly taken up by the dopaminergic
system in the basal ganglia, causing short-term cerebral activation. The
decreases in dopamine receptors can be long lasting and can result in disrupting
the orbital frontal system. Over time, amphetamine and cocaine abusers can
show multiple perfusion defects across both hemispheres of the brain. These
25 effects can be acute and/or chronic.

Traumatic Head Injury

Self-medication with alcohol and drugs can be profound in patients affected by head trauma or closed head injury. Neurological symptoms caused by head trauma are not generally recognized and may present themselves only gradually. Mild injuries to the head often are forgotten especially when they happen in childhood. A child can become difficult or aggressive from a head injury very gradually. The problem may not become apparent until the child becomes old enough to obtain drugs and alcohol with which to self-medicate. Rarely is such behavior attributed to the earlier head injury. Also, rarely can the addicted patient remain sober when the brain trauma goes untreated. The problem that drove the person to use drugs, alcohol, carbohydrates or seek reward in compulsive gambling, risk taking or other activities is still present, and eventually can cause the patient to return to the undesired behavior.

Methods of this invention encompasses the identification of a predisposing factor toward violent behavior or other neurobehavioral disorder by SPECT scan. The anger and aggression that the person feels who has a predisposing factor toward violence may not decrease after withdrawal from addictive substances or behaviors. The anger and aggression may, and can be expected to intensify. With the treatments described herein, the aggression can subside, and unexpectedly, the patient did not relapse into former drug and alcohol use. Based on prior art methods, the methods of this invention would likely have only decreased craving. However, unexpectedly, a combination therapy can reduce aggression or symptoms of withdrawal, can achieve an improved rate of recovery, retain the high rate of recovery over a long period of time. Treatment of these conditions without restoration of neurological functioning can leave the patient vulnerable to outbursts that could be life threatening or result in poor judgement that could reduce the chances for recovery or continued treatment.

Diagnosis Using SPECT Scans

Interpretation of the SPECT scans will be similar to but not limited to the following observations of patterns frequently found in neurological disorders and a sampling of medications that can be useful. In general a SPECT scan follows
5 the administration of a radiolabeled substance that can permit viewing of the brain.

1. Decreased perfusion of anterior prefrontal and inferior orbital prefrontal cortex.

Decreased activity in the prefrontal cortex during a concentration task is often
10 associated with impulsivity, short attention span, distractability and difficulties with organization and planning. There is an association between this finding and ADHD and ADD, especially when this occurs during the performance of a concentration task. This pattern is often responsive to psychostimulant stimulant medication, using, by way of example, Ritalin TM or Formula I of this invention.

15 If decreased activity in the prefrontal cortex is seen during a resting state, it is often associated with depressive disorders, and may be responsive to antidepressant medication, including by way of example only, tricyclic antidepressants. When decreased activity in the prefrontal cortex is seen in both the resting and concentration states, there can be a combination of depression
20 and ADD or ADHD present. This pattern can be observed in patients having head injuries affecting this part of the brain, and later in life in some dementia processes. Formula I can be desirable to treat these patients..

2. Decreased activity in the left lateral frontal area and anterior left temporal lobe can be associated with expressive language disorders. Expressive language
25 disorders can also be caused by head injuries to these parts of the brain. If expressive language disorders are found to be associated with severe mood swings and/or aggressive behavior therapy can be often helped with anti-convulsant medication. Formula II can be desirable to treat these patients.

3. Increased activity anterior cingulate gyrus and medial frontal areas can be associated with problems of shifting attention, which may be clinically manifested by cognitive inflexibility, obsessive thoughts, compulsive behaviors, excessive worrying, argumentativeness, oppositional behavior or “getting stuck” on certain thoughts or actions. There is an especially strong association with this finding and obsessive-compulsive disorders, oppositional defiant disorders, eating disorders, addictive disorders, anxiety disorders (especially when combined with increased basal ganglia activity), Gilles de la Tourette's Syndrome and chronic pain. If clinically indicated, it may be helped by anti-obsessive antidepressants that increase serotonin in the brain. Formulas I and/or III can be useful for these patients.
4. Focal increased activity lateral frontal or prefrontal can be associated with ADHD or ADD or with seizure disorders. Formula II can be useful for these patients.
5. Abnormal activity left temporal lobe (either increased or decreased) can be associated with mood instability, irritability, memory struggles, abnormal perceptions (auditory or visual illusions, periods of deja vu), periods of anxiety with little provocation, periods of spaciness or confusion, and unexplained headaches or abdominal pain. When clinically indicated, abnormalities in this part of the brain can be helped with anticonvulsant medication. Decreased activity in the posterior aspects of the left temporal lobes can be associated with learning problems, especially reading comprehension difficulties and auditory processing problems.
6. Abnormal activity in the right temporal lobe (either increased or decreased; termed herein “right-sided problems”) can be associated with social withdrawal, social skill struggles and depression (more inwardly directed difficulties as opposed to problems with the left side of the brain; herein termed “left-sided problems”). If clinically indicated, these symptoms may be helped by

anti-convulsant medications. When depression is present imipramine can be helpful.

7. Increased basal ganglia activity can be associated with anxiety. Left-sided problems can be associated with irritability, right-sided problems can be associated with inwardly directed anxiety. Formula III can be desirable in patients with this finding. If the finding is focal in nature (more one side than the other), Formula III can also be useful.

8. Diffuse and focal increased limbic system activity can be associated with depression, dysthymia and negativity. Problems left-sided problems can be associated with anger and irritability; right-sided problems can be associated with inwardly directed sadness. Diffuse limbic overactivity tends to be more consistent with depression, whereas focal increased limbic activity (more on one side than the other) is more consistently associated with cyclic mood disorders. Increased focal uptake of the labeled neurochemical in conjunction with patchy increased uptake of that neurochemical across the cortical surface can be associated with cyclothymic or bipolar disorder. If clinically indicated, diffuse increased limbic uptake can be helped by Formula III. If there is also increased activity in the anterior cingulate gyrus, Formula II can be desirable. If there is no or little increase in activity in the anterior cingulate gyrus, one can consider using either Formula II or Formula III.

9. Parietal lobe increased activity in the parietal lobe can be associated with hypersensitivity to noise, touch and taste. The parietal lobes have also been implicated in attentional disorders. In addition, in situations in which there is increased bilateral parietal lobe and lateral frontal lobe activity, a Syndrome termed the "ring of fire" can be observed. It can be desirable to treat patients experiencing this pattern using Formula I.

10. Patchy decreased or increased uptake across the cerebral cortex can be associated with toxic exposure or oxygen deprivation at some point in the past. It

can also be associated with a prior brain infection, drug exposure or widespread trauma. Patchy increased uptake across the cortex is often associated with cyclic mood disorders. This pattern observed using SPECT can be treated using Formula II.

5

11. Focal areas of increased perfusion can be associated with ictal seizure activity or prior head trauma. These patterns can be helped by Formula II.

12. Overall decreased cerebral perfusion can be associated with depression, toxic brain exposure and response to certain medications. Nicotine and large doses of caffeine have been associated with overall decreased cerebral perfusion. Formula III can be desirably used to treat patients having reduced perfusion.

10

13. The "Ring of Fire" pattern includes increased activity in the left and right lateral parietal and frontal lobes and often left and right lateral temporal lobes as well. Often intense increased cingulate activity is also present. This finding can be associated with mood dyscontrol, oppositional behavior, aggressiveness and irritability. It may also be related to bipolar disorder and can be treated using Formula II.

15

Diagnosis by Quantitative Electroencephalography

20 Quantitative electroencephalographic methods (qEEG) can be useful in the diagnosis of underlying neurological disorders or brain trauma. Quantitative EEG relies upon the measurement, using digital technology, of electrical patterns at the surface of the scalp that primarily reflect cortical electrical activity or "brainwaves." The qEEG can be used to detect abnormalities in electrical relationships in various areas of the brain by first comparing the qEEG recording while a patient's eyes are closed with a normative database. A recording of the electrical patterns of the brain is then made with the patient's eyes open and the patient challenged by reading, listening, doing math or other activities, and a

25

comparison of the patient's patterns to a normative database is made. The electrical signals from the patient's brain are captured using a skullcap or a band with a series of electrodes, such as by way of example only, 24. The electrodes monitor the frequency of EEG activity underlying them. It can be particularly
5 useful to evaluate the P300 waves obtained, because alterations in the P300 pattern can be diagnostic of certain neurobehavioral disorders.

Any suitable device known in the field can be used to record the brain waves. Alternatively, it can be desirable to record the electrical activity in the home, to define key areas of the brain most important in identifying neurological
10 conditions most important to treat for long term success in that patient. The frequency is transmitted digitally to a recording device or computer.

In certain embodiments, a useful method includes a recording device and scanning cap or band that has been provided to the patient by mail. The patient places the cap or band on his head, performs the tasks while the recording device
15 receives the data. The recording device is then returned to the clinician, and an analysis is made of the data, and the results of the study can then be faxed, phoned or e-mailed to the patient or the physician. Another method is to have the patient perform the tasks while the data is transmitted in real time directly to a computer or indirectly through the recording device. The data are then
20 analyzed and the results can be returned within moments to the patient or the physician via the computer or the Internet.

Methods of Diagnosis by Infra-Red Spectrophotometry

Diagnosis of underlying neurological disorders or brain trauma using
25 infrared spectroscopy detects changes in oxygenation of the cerebral cortex. Light can penetrate the scalp and skull and is reflected and back-scattered to the surface. The returning light is measured with a photoelectric cell. Oxygenated tissue is red, whereas deoxygenated tissue is blue. The color of the returned light

shows the degree of blood oxygenation. Blood oxygenation is an indicator of volume of blood flowing in a given area of the brain which in turn can correlate with the function of the brain in that area.

5 There can be a correlation between regional cerebral blood flow and cerebral metabolism. Infrared spectroscopy can direct light to those area of the brain which are critical to determine if there are underlying disorders or injury which might be responsible for a patient's need to self-medicate and/or relapse into self-medication with improper or life threatening substances.

10 The method of training the light into the skull can be either a single light source or any number of light sources. The light source and photoelectric cells can be stationary and part of a device that is placed on the head or in a headband or cap. Alternatively, the light source and the photoelectric cells can be movable, either manually by the hand or by mechanical device or computer. Computerized movement can be desirable for accurate comparison to normals and statistical
15 analysis of data.

A scanning device may be simple enough to be used by the patient in the home. Such a device can be sent to the patient's home, the scan done by the patient and the recording device returned for analysis. The results of the analysis can either be faxed, phoned or e-mailed to the patient and/or to his or her
20 physician. These diagnostic methods envision both a resting and/or eyes closed scan and a scan challenging the patient to perform mental tasks. The data in the recording device may also be downloaded to the patient's own computer and transmitted remotely. The patient can receive his or her analysis in real time by e-mail. A specialized database reflecting readings from patients without
25 underlying neurological disorders or brain trauma can provide bases for yielding correlations between an individual patient's function and those of a reference group. The information obtained can be used to obtain results more quickly and less expensively than other brain scanning devices and other methods of

visualizing brain structure or function such as MRI, CAT, FMRI, EEG, qEEG and SPECT. Infrared spectroscopy can be done without invasive procedures, x-ray exposure, dyes or radioactive isotopes. A training device with a single infrared sensor can enable the patient to train and practice at home with only the device and a home computer.

Methods of Diagnosis by Functional MRI

Diagnosis by functional magnetic resonance imaging (MRI) can be used to determine the presence of brain trauma, underlying neurobehavioral disorders by viewing indicators of such disorders by change in the magnetic resonance of tissue and/or blood. In certain embodiments, a desirable method is to combine these tests with the intravenous infusion method, transdermal patch delivery system and/or oral supplementation.

Methods of Diagnosis by MRI, CAT Scan and EEG

Diagnosis using MRI, CAT scans and EEG methods involve using technologies well known in the medical field to diagnose underlying neurological disorders or brain trauma. These methods can use a Magnetic Resonance Imaging (MRI) device, a Computer Assisted Tomography (CAT) device, and/or a standard electroencephalogram (EEG). In certain embodiments, these detection methods can be used to detect changes in brain function before, during and after treating the patients using intravenous infusion, transdermal patch delivery system and/or oral supplementation methods of this invention.

VII. Integration of Diagnosis and Treatment

The methods and compositions of this invention contemplate that a complete history of the patient will be taken to determine the possibility of neurological dysfunction underlying their disorder. When indicated, a “resting”

and a “challenge” SPECT scan will be performed. In certain embodiments, the data can be acquired using a memory device, such as associated with a computer. Data can be transmitted remotely to a physician, who can interpret the results. Physicians trained in neuropharmacology can prescribe appropriate conventional medications where indicated. Such medications will include but not be limited to antidepressant, anticonvulsant, stimulant, or antipsychotic medications. Additionally, it is contemplated that depending upon the diagnosis, a regimen of intravenous compositions of this invention and other adjunct therapies will be instituted. Specialized educational materials are contemplated to be provided to the patient and the patient’s family to assist them in understanding the neurological findings and supporting the patient’s recovery. Subsequent evaluation of the patient’s condition can be taken to determine effectiveness of the medications. Information obtained can be stored in a database on a computer, transmitted remotely to other individuals, using, for example a network, including the internet. Responses can be returned to the patient’s location and thus, the patient’s condition can be monitored during the course of therapy and changes can be made as needed to promote the patient’s recovery.

The other methods described above can also be used to evaluate the course of therapy using the compositions and methods of this invention.

A. Therapy Using Intravenous Ethanol

An alcohol-preferring line of rats exhibit lower serotonergic neurons in the hypothalamus, higher levels of enkephalin in the hypothalamus, more GABA neurons in the nucleus accumbens, reduced dopamine supply at the nucleus accumbens, and reduced dopamine D2 receptor densities. A four-part cascade sequence can occur which can lead to a reduction of dopamine release in the area of the brain believed to be necessary to experience feelings of well-being, i.e., the “reward area.” Rats were then with substances that increase the serotonin in

serotonergic synapses, or stimulate dopamine D2 receptors directly. As a result, the rat's preference for alcohol can diminish.

Although the mechanism for the above phenomenon is not known with certainty, one theory is that by-products of ethanol metabolism that enter the brain via the blood stream are neurotoxic. With abstinence, the individual can go into a withdrawal that can be severe and can include seizures and other life threatening events. By infusing ethanol intravenously and then gradually decreasing the amount of ethanol delivered, serious withdrawal symptoms can be avoided.

Thus, in persons addicted to alcohol, neurotoxic by-products of ethanol can circulate in the blood stream. By-products such as tetrahydroisoquinolines ("TIQ's") can reach the brain where they may play a role in craving. If the individual suffers from leaky-gut syndrome, food and chemical antigens can escape into the blood stream. According to this theory, antibodies to these antigens or to the ethanol or to the ethanol by-products can be circulate in the blood stream and can enter the brain as well, and can contribute to behavioral disorders.

Thus, according to this theory, elimination of alcohol-derived neurotoxins thus can cause the patient to feel better immediately and craving and other compulsions to abate when alcohol is no longer processed through the gut. As the leaky gut heals over several weeks, with the elimination of the ethanol, there is less chance of relapse. The amino acid, glutamate, discussed herein, can be administered to aid in the tightening and closing of the perforations in the gut, aiding healing.

Further, according to another theory, antibodies to ethanol can either bind to or damage dopamine receptor sites in the brain, reducing the availability of available dopamine receptors. Alternatively, an antibody response to a viral antigen can act in the same fashion, either attaching to the receptor thereby

competing with other substances for the receptor site and, or, directly damaging the cell. A feedback mechanism can then gradually and even permanently reduce the dopamine receptor density and thereby interfere in the cascade sequence required to provide the necessary neurotransmitters into the cell. The dopamine
5 or other neurotransmitters needed by brain cells, then can have to compete for the receptor sites. Intravenous infusion of ethanol can either stimulate the dopaminergic system directly or can more successfully compete for the receptor sites with the antibodies that may be present, or with other competing substances, false neurotransmitters or other mimics.

10 The above theories may account for the phenomena of alcohol-induced neurobehavioral disorders. However, other theories may account for the findings, and this invention does not require any particular theory or theories for operability.

To treat alcohol addiction according to certain methods of this invention
15 one can administer ethanol intravenously, in a regimen providing for decreasing dosages of ethanol over a period of ten days. This may be done in the same infusion with "amino acid" formulas discussed herein or be administered by itself or in combination with other approaches described herein, especially anti-craving compounds and pharmaceuticals such as, but not limited to, naloxone,
20 acamprosate, ibogaine, lithium, magnesium chloride, Valium, olanzapine, antitussives such as dextromethorphan and bromotryptoline.

VIII. Genetic and Metabolic Testing to Aid in Treatment

Another method for providing guidance in therapy for Neurobehavioral
25 disorders is to test for genetic indicators of specific Neurobehavioral disorders and to administer by intravenous infusion specialized compounds, the teachings of which are hereby incorporated by reference thereto the entirety of US Patent

No: 5,189,064, US Patent No: 5,210,016, US Patent 4,761,429, and Canadian Patent No: 1,321,146.

Genetic testing and other testing can include laboratory analysis for other metabolic disorders. Specifically, genetic tests for the dopaminergic, adrenergic, serotonergic, cannabinoid receptors as described herein can be performed. Additionally, metabolic evaluation, for example, by Great Smokies Diagnostic Laboratory, and/or others, can provide tests that are diagnostic for: thyroid deficiencies; amino acid deficiencies; amino acid transport disorders; nutritional and metabolic status; various toxicities such as elemental, organophosphate, halogenated hydrocarbon, and other organic xenobiotic toxicities; and an individual's ability to detoxify adverse chemicals via various major metabolic pathways. This testing can aid in determining underlying conditions that require medical treatment in conjunction with or prior to other methods described herein.

Testing can be used as an adjunct to treat an invading virus or the antibody response to a virus that may or may not still be present in the body. The individual may have contracted the virus from the mother in utero or may be genetically susceptible to it and contract it after birth or later in life.

Laboratory tests can be run to determine presence of Epstein-Barr Virus, Cytomegalovirus, Cocksackie Virus, Herpes Viruses, animal viruses, Poliovirus and others to determine if a pattern of viral antigens or sensitivity to any family of viruses is present.

Treatments can include the administration of: known anti-viral pharmaceuticals or pharmaceutical "cocktails;" administration of a vaccine developed by combining all known viruses or families of viruses, particularly the family of enteroviruses, and developing a universal vaccine to the combination; identification of a new virus and development of a vaccine to the new virus; identification of enzyme-destroying anaerobic bacteria in the mouth and treating accordingly; increase the levels of arginine in the brain by dietary and supplement

means; administer the digestive enzymes, trypsin and chymotrypsin in dosages of about 800 to 1,600 mg daily, between meals; administration colloidal silver; administration of autohemotherapeutic ozone; administration of the anti-viral oregano oil; administration of Sanum remedies; and all other such substances
5 reputed to have anti-viral properties.

IX. Method of Providing Medical Advice, Counseling and Support

The final method can include providing the client with his or her own computer, if needed, and, for a period of time, a video connection to the Internet
10 as part of the treatment program and cost. The patient receives CD-ROM's with education and instruction and signs on to the Internet to meet one-on-one with his counselor, his doctor and with other patients in organized support groups. The computer will be supplied in the treatment of Neurobehavioral disorder that has been chronic and where the patient and the family will benefit from medical
15 assistance, counseling and supportive associations with other patients.

A computer system can include means for data acquisition, analysis, storage, transmission to a remote site (such as that of a physician), and can include means for data retrieval from a remote database, storage of remotely acquired data, correlation of clinical, genetic, and metabolic findings, and can
20 provide output locally with the patient to improve the efficacy of therapy.

Industrial Applicability

The compositions and methods of this invention can provide improvement in symptoms of neurobehavioral disorders.

We claim:

1. A composition for treating neurobehavioral disorders, comprising:
 - (a) at least one amino acid;
 - 5 (b) vitamin C; and
 - (c) an electrolyte solution, wherein said composition is sterile.
2. A composition for treating neurobehavioral disorders, comprising:
 - (a) at least one amino acid;
 - 10 (b) a corticosteroid; and
 - (c) an electrolyte solution, wherein said composition is sterile.
3. A composition for treating neurobehavioral disorders, comprising:
 - (a) vitamin C;
 - 15 (b) a corticosteroid; and
 - (c) an electrolyte solution, wherein said composition is sterile.
4. A composition for treating neurobehavioral disorders, comprising:
 - (a) at least one amino acid;
 - 20 (b) an immune potentiating amount of gamma-globulin; and
 - (c) an electrolyte solution, wherein said composition is sterile.
5. A composition for treating neurobehavioral disorders, comprising:
 - (a) at least one amino acid;
 - 25 (b) an inhibitor of opioid peptide degradation sufficient to inhibit degradation of opioid peptides; and
 - (c) an electrolyte solution, wherein said composition is sterile.

6. The composition of claim 1, further comprising:
a neurotransmitter synthesis-promoting amount of at least one neurotransmitter precursor selected from the group consisting of dopamine precursors, serotonin precursors, or GABA precursors.

5

7. The composition of claim 6, wherein said dopamine precursor is selected from the group consisting of L-tyrosine, L-phenylalanine and L-DOPA.

8. The composition of claim 6, wherein said serotonin precursor is selected from the group consisting of L-tryptophan and 5-hydroxytryptophan.

9. The composition of claim 6, wherein said GABA precursor is selected from the group consisting of L-glutamine, L-glutamic acid and L-glutamate.

10. The composition of claim 5, wherein said inhibitor of opioid degradation is selected from the group consisting of amino acids, peptides and structural analogues or derivatives thereof.

11. The composition of claim 5, wherein said inhibitor is D-phenylalanine.

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12. The composition of claim 1, further comprising
a tryptophan concentration enhancing amount of chromium picolinate or chromium nicotinate.

13. The composition of claim 12, further comprising a chromium utilization enhancing amount of vitamin B-6.

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14. The composition of claim 13, wherein said vitamin B-6 is pyridoxal-5'-phosphate.

15. The composition of claim 1, further comprising an amino acid absorption and utilization enhancing amount of alpha-keto-glutarate.

16. The composition of claim 1, further comprising a neurotransmitter synthesis promoting amount of oxytocin.

17. The composition of claim 1, further comprising an opiate receptor antagonist sufficient to inhibit the effects of an opiate on a receptor for said opiate.

18. The composition of claim 1, further comprising a dopamine receptor agonist sufficient to activate post-synaptic dopamine receptors in the striatum.

19. The composition of claim 18, wherein said dopamine receptor agonist is selected from the group consisting of bromocryptine and aacomprosate

20. The composition of claim 1, wherein said at least one amino acid is selected from the group consisting of L-taurine, L-tyrosine, L-phenylalanine, L-glutamine, D-phenylalanine, L-tryptophan, 5-hydroxytryptophan, arginine, methionine, leucine, isoleucine, histidine, valine, glycine, serine, asparagine, aspartic acid, citrulline, glutamic acid, ornithine and homocysteine.

21. The composition of claim 20, wherein said at least one amino acid is present at a dose of between about 1 µg and about 100 mg each.

22. The composition of claim 1, further comprising an amount of ammonia scavenger sufficient to prevent neurotoxic effects of ammonia generated by said at least one amino acid.

5 23. The composition of claim 1, further comprising a brain seizure inhibiting amount of taurine or L-GABA.

24. The composition of claim 22, wherein said ammonia scavenger is selected from the group consisting of citric acid, alpha-keto-glutarate, magnesium,
10 aspartic acid and glutamic acid.

25. The composition of claim 1, further comprising an antiinflammatory agent.

15 26. The composition of claim 25, wherein said antiinflammatory agent is selected from the group consisting of corticosteroids, vitamin C and L-glutamate.

27. The composition of claim 26, wherein said corticosteroid is derived from adrenal cortical extract.

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28. The composition of claim 4, wherein said gamma globulin is present in an amount in the range of about 5 grams to about 10 grams.

29. The composition of claim 1, further comprising at least one vitamin
25 selected from the group consisting of niacin, niacinamide, niacinamide ascorbate, vitamin B-complex, vitamin B-12, vitamin C, riboflavin, riboflavin-5-phosphate, N-acetylcysteine, folic acid, folacin, vitamin E, biotin, vitamin A, pantothenic acid,

coenzyme Q10, thiamin, beta-carotene, para-aminobenzoic acid, bioflavonoids, and pyridoxine HCl.

30. The composition of claim 1, further comprising an insulin potentiating
5 amount of a substance selected from the group consisting of iron aspartate/chelate, magnesium oxide, zinc chelate, selenium, vanadium, inositol, thiamin mononitrate, thiamin HCl, vitamin A, vitamin C, bioflavonoids, para-amino benzoic acid, and pyridoxine HCl.

10 31. The composition of claim 1, further comprising an amino acid metabolism promoter selected from the group selected from zinc aspartate/picolinate/chelate, copper, chromium, magnesium, manganese, selenium, potassium, calcium, and sodium.

15 32. An oral composition for treating neurobehavioral disorders, comprising at least one amino acid and a substance selected from the group consisting of Ginkgo Biloba, methylsulfonylmethane, phosphatidylserine, phosphatidylcholine, alpha lipoic acid, red ginseng root, L-aspartic acid, ephedrine, pancreatic enzymes, caffeine, theobromine, Hypericum perforatum extract, S-adenosyl methionine,
20 dihydroxyacetate, DMAE, grape seed extract, betaine, prickly pear cactus extract, Gymnea sylvestre extract, nicotinamide adenine dinucleotide/hydrogen, cholecystokinin, Cyclo (His-Pro), corticotropin-releasing hormone, neuropeptide Y, galanin, monolaurin and fructo-oligosaccharides.

25 33. A method for treating a subject having an neurobehavioral disorder, comprising the step of administering intravenously, a composition comprising at least one amino acid.

34. A method for treating a subject having an neurobehavioral disorder, comprising

administering intravenously to said subject a composition comprising

- (a) vitamin C;
- 5 (b) a corticosteroid; and
- (c) water, wherein said composition is sterile and is isotonic.

35. A method for treating a subject having an neurobehavioral disorder, comprising

10 administering intravenously to said subject a composition comprising

- (a) at least one amino acid;
- (b) an immune potentiating amount of gamma-globulin; and
- (c) water, wherein said composition is sterile and is isotonic.

15 36. A method for treating a subject having an neurobehavioral disorder, comprising

administering intravenously to said subject a composition comprising

- (a) at least one amino acid;
- (b) an inhibitor of opioid peptide degradation sufficient to inhibit
- 20 degradation of opioid peptides; and
- (c) water, wherein said composition is sterile and is isotonic.

37. The method of claim 33, further comprising the step of:

injecting intravenously a neurotransmitter synthesis-promoting amount of at
25 least one neurotransmitter precursor selected from the group consisting of
dopamine precursors, serotonin precursors, or GABA precursors.

38. The method of claim 37, wherein said dopamine precursor is selected from the group consisting of L-tyrosine, L-phenylalanine and L-DOPA.

39. The method of claim 37, wherein said serotonin precursor is selected from
5 the group consisting of L-tryptophan and 5-hydroxytryptophan.

40. The method of claim 37, wherein said GABA precursor is selected from the group consisting of L-glutamine, L-glutamic acid and L-glutamate.

10 41. The method of claim 36, wherein said inhibitor of opioid degradation is selected from the group consisting of amino acids, peptides and structural analogues or derivatives thereof.

42. The method of claim 36, wherein said inhibitor is D-phenylalanine.
15

43. The method of claim 33, further comprising intravenously injecting a tryptophan concentration enhancing amount of chromium picolinate or chromium nicotinate.

20 44. The method of claim 43, further comprising intravenously injecting a chromium utilization enhancing amount of vitamin B-6.

45. The method of claim 44, wherein said vitamin B-6 is pyridoxal-5'-phosphate.
25

46. The method of claim 33, further comprising an amino acid absorption and utilization enhancing amount of alpha-keto-glutarate.

47. The method of claim 33, further comprising injecting intravenously a neurotransmitter synthesis promoting amount of Rhodila or nubazine.

48. The method of claim 33, further comprising injecting intravenously an
5 opiate receptor antagonist sufficient to inhibit the effects of an opiate on a receptor for said opiate.

49. The method of claim 33, further comprising injecting intravenously a
10 dopamine receptor agonist sufficient to activate post-synaptic dopamine receptors in the striatum.

50. The method of claim 49, wherein said dopamine receptor agonist is selected from the group consisting of bromocryptine and Acomprosate™

15 51. The method of claim 33, wherein said at least one amino acid is selected from the group consisting of L-taurine, L-tyrosine, L-phenylalanine, L-glutamine, D-phenylalanine, L-tryptophan, 5-hydroxytryptophan, arginine, methionine, leucine, isoleucine, histidine, valine, glycine, serine, asparagine, aspartic acid, citrulline, glutamic acid, ornithine and homocysteine.

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52. The method of claim 49, wherein said at least one amino acid is present at a dose of between about 1 µg and about 100 mg each.

25 53. The method of claim 33, further comprising injecting intravenously an amount of ammonia scavenger sufficient to prevent neurotoxic effects of ammonia generated by said at least one amino acid.

54. The method of claim 33, further comprising injecting intravenously a brain seizure inhibiting amount of taurine or L-GABA.

55. The method of claim 53, wherein said ammonia scavenger is selected from the group consisting of citric acid, alpha-keto-glutarate, magnesium, aspartic acid and glutamic acid.

56. The method of claim 33, further comprising injecting intravenously an antiinflammatory agent.

57. The method of claim 56, wherein said antiinflammatory agent is selected from the group consisting of corticosteroids, vitamin C and L-glutamate.

58. The method of claim 33, wherein said gamma globulin is present in an amount in the range of about 5 grams to about 10 grams.

59. The method of claim 33, further comprising at least one vitamin selected from the group consisting of niacin, niacinamide, niacinamide ascorbate, vitamin B-complex, vitamin B-12, vitamin C, riboflavin, riboflavin-5-phosphate, N-acetylcysteine, folic acid, folacin, vitamin E, biotin, vitamin A, pantothenic acid, coenzyme Q10, thiamin, beta-carotene, para-aminobenzoic acid, bioflavonoids, and pyridoxine HCl.

60. The method of claim 33, further comprising an insulin potentiating amount of a substance selected from the group consisting of iron aspartate/chelate, magnesium oxide, zinc chelate, selenium, vanadium, inositol, thiamin mononitrate, thiamin HCl, vitamin A, vitamin C, bioflavonoids, para-amino benzoic acid, and pyridoxine HCl.

61. The method of claim 33, further comprising an amino acid metabolism promoter selected from the group selected from zinc aspartate/picolinate/chelate, copper, chromium, magnesium, manganese, selenium, potassium, calcium, and sodium.

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62. A method for treating a patient having a neurobehavioral disorder, comprising the steps of:

(a) evaluating a neurobiological characteristic of said neurobehavioral disorder; and

10 (b) injecting into said patient, an intravenous composition sufficient to treat said Neurobehavioral disorder.

63. The method of claim 62, wherein said step of evaluating is carried out using a method selected from single positron emission computed tomography (SPECT),
15 quantitative electroencephalography (qEEG), infrared spectrophotometry (IR), magnetic resonance imaging (MRI), and computer assisted tomography (CAT).

64. The method of claim 62, further comprising the step of reevaluating said neurobiological characteristic of said neurobehavioral disorder.

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65. A method of treating at least one symptom of traumatic brain injury, comprising:

(a) evaluating a neurobiological characteristic of said traumatic brain injury; and

25 (b) injecting intravenously into said patient, an intravenous composition in an amount sufficient to treat said symptom.

66. A method of treating a patient having an neurobehavioral disorder, comprising the steps of:

(a) evaluating a neurobiological characteristic of said neurobehavioral disorder; and

5 (b) injecting intravenously into said patient a sufficient amount of a composition that reverses said neurobiological characteristic.

67. The method of claim 66, wherein said neurobehavioral disorder is associated with disorders of the adrenergic system, wherein said composition
10 comprises:

(a) S-adenosyl methionine;

(b) D-phenylalanine;

(c) L-phenylalanine;

(d) DL-phenylalanine;

15 (e) tyrosine;

(f) DHEA; and

(g) Siberian ginseng, and wherein said composition is administered orally..

20 68. The method of claim 67, further comprising the step of administering at least one substance selected from the group consisting of methylphenidate, L-methionine, adenosine, vitamin B-6, vitamin B-12, magnesium, DMG betaine, TMG and folic acid.

25 69. The method of claim 66, wherein said neurobehavioral disorder includes depression, and wherein said composition comprises:

(a) TMG-betaine;

(b) at least one amino acid from the group consisting of

L-methionine, L-glutamine, L-glycine and L-cysteine;

- (c) folic acid;
- (d) calcium;
- (e) vitamin B-12;
- 5 (f) magnesium;
- (g) N-acetylcysteine;
- (h) adenosine triphosphate;
- (i) zinc aspartate;
- (j) magnesium aspartate; and
- 10 (k) vitamin B-6.

70. The method of claim 60, further comprising the step of orally administering said composition.

15 71. A method for treating a patient with alcohol addiction, comprising the steps of:

(a) providing a first treatment on a first day, said treatment comprising intravenous injection of a solution of ethanol in the range of about 5% to about 25% by volume for a total volume of about 220 ml ;

20 (b) providing subsequent intravenous treatments on subsequent days with progressively lower amounts of ethanol until symptoms improve.

72. A method of treating a patient having an neurobehavioral disorder, comprising the steps of:

- 25 (a) evaluating said patient's symptoms to provide data correlating said symptoms to said patient's disorder;
- (b) storing said data on a storage device;
- (c) retrieving said data from said storage device;

(d) treating said patient, said treatment comprising:

(i) injecting intravenously, at least one of an amino acid, a vitamin, a neurotransmitter precursor, an inhibitor of opiate degradation, an immune system enhancer and a corticosteroid; and

5 (e) reevaluating said patient's symptoms to provide data correlating said symptoms to said patient's treatment.

73. The method of claim 72, wherein said data is selected from the group consisting of SPECT, qEEG, IR spectrophotometry, patient history and analysis of
10 genetic alleles.

74. The method of claim 72, further comprising the step (f) of storing the data obtained in step (e) on a storage device.

15 75. The method of claim 72, further comprising the step of retrieving said data stored in step (f).

76. The method of claim 74, further comprising the step of (g) correlating the data obtained in step (a) with the data obtained in step (e).
20

77. The method of claim 76, further comprising the step (h) of providing correlated data obtained in step (g) to one of a health care practitioner and the patient.

25 78. The method of claim 76, wherein said storage device is associated with a computer.

79. The method of claim 76, wherein said step (g) is carried out using a computer.

80. The method of claim 77, wherein said step (h) is carried out using a means
5 of remote communication.

81. The method of claim 80, wherein said remote communication is carried out using a computer and a network

10 82. A composition for treating a patient having a neurobehavioral disorder, comprising:

(a) a neurotransmitter synthesis-enhancing amount of at least one neurotransmitter precursor;

(b) an effective amount of an inhibitor of opioid degradation;

15 (c) a substance selected from the group consisting of thymus extract, L-aurine, alpha-keto glutarate, lidocaine, L-glutathione, pyridoxal-5-phosphate, sodium ascorbate, oxytocin, L-glycine, L-leucine, gamma globulin, vitamin B complex, magnesium taurate, citric acid, chromium polynicotinate, chromium nicotinate, chromium picolinate, zinc chelate, calcium chelate, vitamin B-12,
20 vitamin B-5, vitamin B-6, vitamin B-1, folic acid, L-aurine and balanced amino acid solution with electrolytes.

83. The composition of claim 82, wherein said neurotransmitter precursor is selected from the group consisting of L-tyrosine, L-phenylalanine, L-DOPA, L-tryptophan, 5-hydroxytryptophan, L-glutamine, L-glutamic acid and L-glutamate.
25

84. The composition of claim 82, wherein said inhibitor of opioid degradation is D-phenylalanine.

86

85. A composition for treating a patient with a neurobehavioral disorder, comprising:

- (a) an effective amount of an inhibitor of opioid degradation; and
- (b) a substance selected from the group consisting of thymus extract,
5 L-taurine, alpha-keto glutarate, lidocaine, L-glutathione, pyridoxal-5-phosphate, sodium ascorbate, oxytocin, L-glycine, L-leucine, gamma globulin, vitamin B complex, magnesium taurate, citric acid, chromium polynicotinate, chromium nicotinate, chromium picolynate, zinc chelate, calcium chelate, vitamin B-12, vitamin B-5, vitamin B-6, vitamin B-1, folic acid, L-taurine and balanced amino
10 acid solution with electrolytes.

86. A method for treating a patient having a neurobehavioral disorder, comprising the steps of:

- (a) diagnosing said patients neurobehavioral disorder;
- 15 (b) selecting a composition of claim 84 effective against said disorder; and
- (c) administering said composition to said patient until symptoms of said disorder are reduced.

20 87. The method of claim 86, wherein said composition is selected from claim 85.

88. A composition for treating the symptoms of a neurobehavioral disorder, comprising:

- 25 (a) an amount of D-phenylalanine effective to reduce said symptoms of said neurobehavioral disorder; and
- (b) a sterile carrier.

89. A composition for treating the symptoms of a neurobehavioral disorder, comprising:

(a) L-glutathione effective to reduce said symptoms of said neurobiological disorder; and

5 (b) a sterile carrier.

90. A composition for treating the symptoms of a neurobehavioral disorder, comprising:

10 (a) an amount of a D-amino acid effective to reduce said symptoms of said neurobehavioral disorder; and

(b) a sterile carrier.

91. A composition for treating the symptoms of a neurobehavioral disorder, comprising:

15 (a) at least one amino acid in an amount effective to reduce said symptoms of said neurobehavioral disorder;

(b) D-phenylalanine in an amount sufficient to inhibit degradation of an opioid; and

20 (c) at least one substance selected from the group consisting of thymus extract, L-aurine, alpha-keto glutarate, lidocaine, L-glutathione, pyridoxal-5-phosphate, sodium ascorbate, oxytocin, L-glycine, L-leucine, gamma globulin, vitamin B complex, magnesium taurate, citric acid, chromium polynicotinate, chromium nicotinate, chromium picolynate, zinc chelate, calcium chelate, vitamin B-12, vitamin B-5, vitamin B-6, vitamin B-1, folic acid, L-aurine and balanced
25 amino acid solution with electrolytes.

92. A composition for treating the symptoms of a neurobehavioral disorder, comprising the following substances in the ranges of amounts indicated:

	L-tyrosine	4000 - 7000 mg
	L-glutamine	2500 - 3000 mg
	L-tryptophan	1000 - 5000 mg
	(or 5HTP)	1000 - 5000 mg
5	L Phenylalanine	3000 - 6000 mg
	D-phenylalanine	3000 - 7000 mg
	L-glutathione	250 - 3000 mg
	Alpha-keto glutarate	50 - 400 mg
	P-5-p	50 - 400 mg
10	Thymus extract	2 - 4 ml
	Lidocaine (1%)	5 - 10 mg
	Na ascorbate	5 - 50 gm
	Cr polynicotinate	8 - 16 µg
	nicotinate or	8 - 16 µg
15	picolynate	8 - 16 µg
	Zn chelate	200 - 1500 mg
	MgO or Mg taurate	500 - 1500 mg
	Ca chelate	500 - 1500 mg
	vitamin B12	0.5 - 3 mg
20	vitamin B12 push	0.5 - 1.5 mg
	vitamin B complex	0.5 - 1.5 mg
	vitamin B5	50 - 150 mg
	vitamin B6	50 - 150 mg
	vitamin B1	50 - 150 mg
25	folic acid	0.03 - 0.09
	oxytocin	1 - 10 ml
	Balanced amino acid solution with electrolytes,	1000 ml

93. A composition for treating symptoms of a neurobehavioral disorder, comprising the following substances in the ranges of amounts indicated:

	L-aurine	150 - 2000 mg
	L-tyrosine	500 - 1500 mg
5	L-glutamine	7000 - 10,000 mg
	L-tryptophan	1000 - 4000 mg
	(or 5HTP)	3000 - 7000 mg
	L Phenylalanine	1000 - 6000 mg
	D-phenylalanine	1000 - 7000 mg
10	L-glutathione	250 - 3000 mg
	Alpha-keto glutarate	50 - 400 mg
	P-5-p	50 - 400 mg
	Thymus extract	2 - 4 ml
	Lidocaine (1%)	5 - 10 mg
15	Na ascorbate	10 - 60 gm
	Cr polynicotinate	8 - 16 µg
	Zn chelate	200 - 1500 mg
	MgO or Mg taurate	500 - 1500 mg
	Ca chelate	1200 - 2500 mg
20	vitamin B12	0.5 - 3 mg
	vitamin B12 push	0.5 - 1.5 mg
	vitamin B complex	0.5 - 1.5 mg
	vitamin B5	50 - 150 mg
	vitamin B6	50 - 150 mg
25	vitamin B1	50 - 150 mg
	folic acid	0.03 - 0.09
	oxytocin	1 - 10 ml
	Balanced amino acid solution with electrolytes,	1000 ml

94. A composition for treating symptoms of a neurobehavioral disorder, comprising the following substances in the ranges of amounts indicated:

	L-aurine	150 - 7000 mg
	L-tyrosine	4000 - 7000 mg
5	L-glutamine	7000 - 10,000 mg
	L-tryptophan	1000 - 4000 mg
	(or 5HTP)	3000 - 7000 mg
	L Phenylalanine	3000 - 6000 mg
	D-phenylalanine	3000 - 7000 mg
10	L-glutathione	250 - 3000 mg
	Alpha-keto glutarate	50 - 400 mg
	P-5-p	50 - 400 mg
	Thymus extract	2 - 4 ml
	Lidocaine (1%)	5 - 10 mg
15	Na ascorbate	10 - 60 gm
	Cr polynicotinate	8 - 16 µg
	Zn chelate	200 - 1500 mg
	MgO or Mg taurate	500 - 1500 mg
	Ca chelate	500 - 1500 mg
20	vitamin B12	0.5 - 3 mg
	vitamin B12 push	0.5 - 1.5 mg
	vitamin B complex	0.5 - 1.5 mg
	vitamin B5	50 - 150 mg
	vitamin B6	50 - 150 mg
25	vitamin B1	50 - 150 mg
	folic acid	0.03 - 0.09
	oxytocin	1 - 10 ml
	L-leucine	300 - 1500 mg

91

Balanced amino acid solution with electrolytes, 1000 ml

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